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ORAL SURGERY

JOURNAL OF ORAL SURGERY

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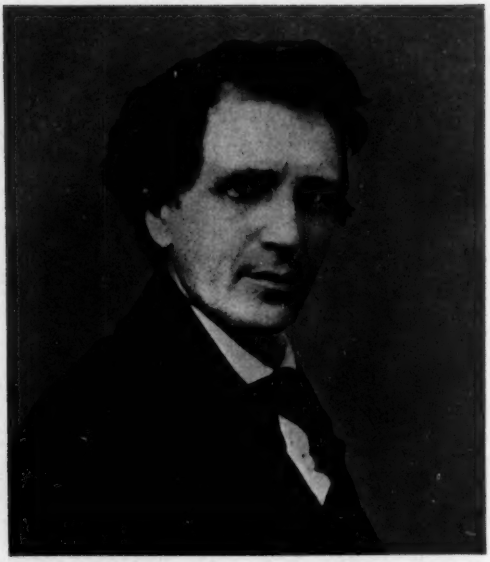

SIMON P. HULLIHEN

Simon P. Hullihen, M.D., D.D.S. (1810-1857), frequently called the "Father of Oral Surgery," was born in Northumberland County, Pennsylvania. His education from early youth was directed toward the practice of medicine. He acquired the degree of Doctor of Medicine from the Washington Medical College, Baltimore, Maryland, in 1832. Soon after entering the practice of medicine in Wheeling, West Virginia, he determined to devote his attention to the practice of dental and oral surgery as a specialty. He attained such prominence in this field that the Baltimore Dental College conferred upon him the degree of Doctor of Dental Surgery in 1843.

Doctor Hullihen developed remarkable skill in what was then a pioneer field of operation—cleft lip and cleft palate. Over a period of some twenty years he operated upon 90 cases of cleft lip, 50 to 60 cases of cleft palate and 100 cases of cancer of the face and mouth. In addition, he performed some 80 plastic operations of the nose, lip and jaw.

Doctor Hullihen foresaw the necessity of an all embracing medical-surgical-dental understanding and by his professional accomplishments contributed much to the founding of the practice of oral surgery as a specialty.

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Surgical management of cancer of the mouth

■ *James B. Smith, D.D.S., M.S., Danville, Pa.*

The treatment of the patient who has cancer of the mouth is often a complex matter of judgment based on a large number of factors. It would seem a simple matter to review the statistical studies in the literature and learn whether surgery, or roentgen therapy, or gamma radiation, or which combination thereof offers the best chance for the cure of the patient. Unfortunately however such a study does not solve the problem for the individual patient but only indicates the treatment of choice for the average patient. No formula has been devised which will consider all the features of tumor, patient and operator, and indicate the best treatment for the individual patient. The final plan of treatment is, therefore, a matter of judgment based on a knowledge of all forms of treatment of the disease. Such knowledge is most likely to be possessed by men trained in both roentgenology and surgery, or by groups of radiologists, surgeons and pathologists working together in tumor conferences usually located in large medical centers.

In view of the foregoing statements it is not desirable to discuss the surgical treatment of cancer of the mouth separately from roentgen therapy and gamma radiation. Therefore some of the indications for, if not the technic of, roentgen therapy will appear in this section.

■ *Treatment planning*

After the diagnosis has been established and a histologic study of a biopsy specimen has been made it is necessary to decide what can be expected of treatment measures. If the patient is relatively young and in good health and if the tumor is small and well differentiated histologically, the most radical measures are justified in an effort to effect a complete cure. In such a clear-cut case many if not all oncologists would advise the surgical removal of the tumor with a wide margin of normal tissue. However, if the patient is old, in poor health and has a large tumor of long duration which is

Read at the annual clinic day of Oral Surgery Department of Henry Ford Hospital.

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highly anaplastic, it would be poor judgment to institute radical surgical measures hoping to cure the patient. In such a case it is often best to admit the hopelessness of the situation and use palliative measures only. Between these two extremes lie many combinations of factors which make every case an individual problem. However, a survey of all the information at hand will usually indicate whether a cure can be hoped for, whether palliative treatment must be used or whether any treatment whatsoever is indicated. It must be pointed out that irradiation therapy is accompanied by a severe local reaction if it is used effectively on the tumor, and that old debilitated people should not be subjected to this treatment unless it is certain that the benefit will outweigh the discomfort produced by the treatment.

For the patients in whom a cure is expected surgical treatment is advisable. The shorter the duration of the disease and the smaller the lesion the more certain is the cure. Irradiation therapy in this hospital is usually reserved (on the advice of our radiologist) for advanced cases wherein surgical treatment is inadvisable because of the extent of the tumor or the poor physical condition of the patient. The combined use of surgical treatment and roentgen therapy or gamma radiation for treatment of the primary mouth cancer is never planned as the treatment of choice. This combined treatment is used only where the surgeon's preoperative judgment was faulty and all of the tumor could not be removed at operation. In such a case it would have been better to spare the patient the risk and expense of the operation. After such an operation the patient must receive as much radiation to destroy the last bit of tumor as would have been necessary to destroy the original tumor. He, therefore, has suffered the undesirable effects of two forms of treatment for one disease. In order to discuss the surgical management of cancer of the mouth typical cases will be presented and the method of treatment outlined.

■ *Cancer of the Lower Lip*

REPORT OF A CASE—On January 6, 1948, a 44 year old, white, anthracite coal miner was referred to the Geisinger Memorial Hospital complaining of a lesion on his lower lip. When first noticed six months previously, the lesion was very small but it steadily increased in size. There had never been any pain associated with the lesion.

Examination revealed an ulcer 1 cm. in diameter in the mucosa of the middle third of the lower lip (Fig. 1, left). The margin of the lesion was elevated and rolled. On palpation the lesion was indurated and nontender. Palpation of the submaxillary and cervical regions failed to reveal any enlarged lymph nodes. Systemic review revealed a chronic morning cough productive of green-black sputum and associated with chest pain. Dyspnea occurred on exertion, but ankle edema was denied.

Physical examination revealed a poor state of oral hygiene. The tonsils were enlarged and inflamed. Breath sounds were accentuated, but no rales were heard. Roentgenograms of the chest revealed regions of increased density in the upper lobes of both lungs. Sputum examination failed to demonstrate acid-fast bacilli. Blood count and urinalysis were

Fig. 1.—Left: Squamous cell carcinoma of the lower lip. Right: Lower lip after wide excision of carcinoma

within normal limits and the Wassermann examination was negative. With the diagnosis of cancer of the lower lip and anthracosilicosis secondary to his occupation, the patient was admitted to the hospital for operation.

On January 7, 1948, a wedged shaped section of the lower lip including a full centimeter margin of normal tissue on either side of the lesion was excised, using 2 per cent procaine hydrochloride infiltration anesthesia. The mucous membrane was approximated with interrupted silk sutures, the muscle layer was approximated with 3-0 chromic gut sutures and the skin was closed with 00000 nylon suture material on an atraumatic needle.

For three days after the operation the patient received 20,000 units of penicillin every three hours for prevention of infection in the wound. The histologic diagnosis of tissue removed at operation was squamous cell carcinoma (grade II according to Broder's index). The patient went home on January 12, 1948, and returned for removal of sutures on the following day. When next seen on February 3, his lip was well healed with only a linear scar to mark the site of the operation (Fig. 1, right).

Careful consideration was given to the question of subsequent operation for removal of the lymph node bearing tissue from the suprahyoid regions bilaterally. However, enlarged nodes could not be palpated, and the patient agreed to appear regularly for observation. Therefore, neck dissection was deferred. This patient was last seen on March 24, 1948, but has failed to reappear for observation though three recall letters have been sent to him.

REPORT OF A CASE—A second patient with cancer of the lip will illustrate a clear-cut indication for roentgen therapy.

On April 23, 1947, a 59 year old silk mill worker was referred to the Oral Surgery Department of the Geisinger Memorial Hospital complaining of a lesion on the lower lip which had been present for approximately one month. He had noticed chapping of the lip for about four months.

Examination revealed a dry crusting lesion in the middle third of the vermillion border of the lower lip approximately .6 by 3 cm. in size. The margins of this lesion were elevated and rolled. On palpation the lesion was indurated and nontender. A small fissure lay in the midline of the lip (Fig. 2, left). Palpation of the submaxillary and cervical regions bilaterally revealed no evidence of enlarged lymph nodes. From these observations the

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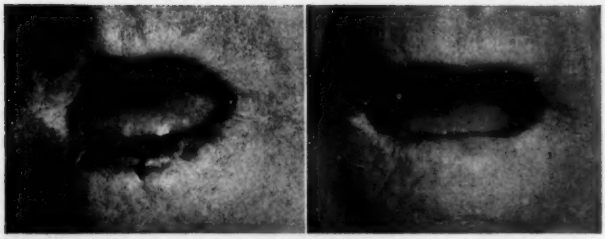

Fig. 2.—Left: Squamous cell carcinoma of the lower lip, before treatment. Right: Lower lip after roentgen therapy. Note loss of hair in irradiated area

clinical diagnosis of carcinoma of the lower lip was made.

On April 24, using 2 per cent procaine hydrochloride anesthesia, a triangular section of tissue measuring 1 cm. along the margin of the lip was removed for microscopic examination. The histologic diagnosis thus obtained was squamous cell carcinoma (grade II according to Broders' index). Blood Wassermann reaction was reported negative.

ROENTGEN THERAPY FOR CANCER OF THE LOWER LIP—Adequate surgical treatment of this tumor involving one half of the lower lip would certainly produce a deformity which would make the patient a social outcast for the rest of his life. This procedure would of course be justified in the treatment of cancer if no other method provided an equal chance of cure. Roentgen therapy, however, if administered in heavy dosage by a radiologist adequately trained in cancer therapy, will produce a five year cure rate comparable with the cure rate for the surgical method of treatment of cancer of the lip. Unfortunately the time required for proper roentgen therapy and the pain suffered by the patient after heavy irradiation of the lip are much greater than would be the case if surgical treatment could be used, but in this case the cosmetic result seemed to outweigh the disadvantages.

A lead shield was constructed to protect the tongue and mandible, and the lip was given eight treatments over a period of 18 days, totaling 4,000 roentgens measured in air (factors: 100 kv., 28 inch distance, 1 mm. aluminum filter, field size 2 by 4 mm.). Figure 2, right, shows the lower lip on April 10, 1948, one year after the last treatment.

This patient has been examined at two month intervals since the completion of his treatment. On his last visit, July 24, 1948, there was no evidence of local recurrence of the tumor or of enlarged lymph nodes. Neck dissection will be deferred indefinitely unless lymph nodes become palpable. This policy of watchful waiting is in accord with McClure and Lam[1] who believe that this is the best course to follow provided the patient will return for careful examination at two month intervals for a period of three to five years. This patient has given up smoking, and keeps his lip coated with a film of petroleum jelly when he is exposed to the sun or wind.

1. McClure, R. D., and Lam, Conrad R., Should the Neck Nodes be Dissected in Patients with Carcinoma of the Lip? *Ann. Surg.* 125:658 (May) 1947.

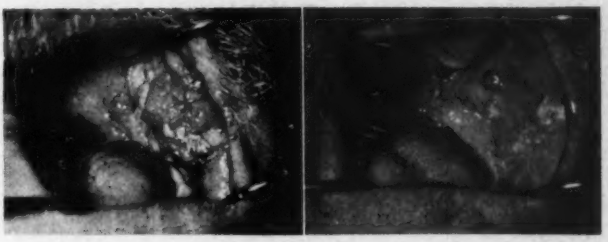

Fig. 3.—Left: Squamous cell carcinoma of left buccal mucosa. Note area of leukoplakia. Right: Buccal mucosa after roentgen therapy. Note persistence of leukoplakia

■ *Cancer of the Buccal Mucosa*

Cancer of the buccal mucosa like cancer of the lip is closely associated with chronic irritation. The habit of chewing tobacco is common among patients who develop this neoplasm, and in many cases tobacco most certainly has been the direct cause of the tumor. In these patients the first lesion to develop consists of an area of leukoplakia in that part of the buccal mucosa which comes in contact with the tobacco. The management of this form of cancer, therefore, begins with the management of leukoplakia. This is a difficult problem in itself, for often a major portion of the oral mucosa is involved. In such cases a conservative type of treatment may be used. All use of tobacco should be discontinued and the lesion should be carefully examined at three month intervals as long as it persists. However, if the lesion tends to pile up and become indurated cauterization of small areas may be indicated. If a major portion of the buccal mucosa is involved, excision of the leukoplakia will have to be followed by application of a split thickness skin graft if scarring and contracture are to be avoided.

Cancer of the buccal mucosa is usually treated by excision of a full thickness section of the cheek. The defect may then be repaired by plastic surgery. Frequently, however, the lesion is too extensive to allow surgical intervention or the patient is too old to stand the operation. In the case discussed next the extent of the lesion and the age of the patient prevented surgical treatment.

REPORT OF A CASE—Figure 3, left, illustrates a squamous cell carcinoma, grade II, involving the entire left buccal mucosa of a 74 year old white man who was referred to the Oral Surgery Department of the Geisinger Memorial Hospital on August 24, 1945. He insisted that the lesion had been present no longer than four or five weeks, and that it had never caused any pain. He volunteered the information that he had chewed tobacco for 61 years and always held the tobacco in the left side of his mouth.

TREATMENT—Surgical treatment for this cancer was thought to be inadvisable because of the patient's age and the large size of the lesion. Therefore, palliative roentgen therapy was instituted. Figure 3, right, shows the buccal mucosa on December 19, 1946. The fungating lesion, shown in Figure 3, left, completely disappeared following heavy roentgen therapy, but areas of leukoplakia persisted.

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He was last seen on September 27, 1947, at which time he had an ulcer approximately 1 cm. in diameter in the left buccal mucosa which probably represented recurrent carcinoma. He refused biopsy. Since he had had the maximum dose of radiation compatible with vitality of the surrounding normal tissue, further roentgen therapy was withheld, and he is being treated symptomatically for relief of pain.

This case not only demonstrates the palliative use of roentgen therapy, but also shows the extreme resistance of leukoplakia to roentgen therapy, for the leukoplakia persisted even though the neoplasm temporarily disappeared.

■ *Cancer of the Tongue*

Cancer of the tongue is usually best treated by surgical intervention, for large sections of the tongue can be removed without serious deformity. Large lesions in old people are often best treated with gamma radiation from radon seeds or radium needles. The lesion illustrated in figure 4, left, is an ideal one for surgical treatment.

REPORT OF A CASE—On October 29, 1947, a 38 year old white man was referred to the Oral Surgery Department of the Geisinger Memorial Hospital complaining of soreness in the right side of his tongue. Two months before the onset of soreness his dentist had pointed out a lesion on the tongue.

EXAMINATION—Examination revealed a white plaque 5 mm. in diameter on the right lateral margin in the middle third of the tongue. Just anterior to this plaque there was an ulcer 3 mm. in diameter (Fig. 4, left). On palpation the base of the ulcer was definitely indurated. With these findings of leukoplakia, ulceration and induration a clinical diagnosis of cancer was made.

The patient was admitted to the hospital for excision of the lesion on October 30 without previous biopsy. At operation a section of tongue 2 by 1.5 by 1 cm. was excised using local anesthesia and sharp dissection. The wound was closed immediately with silk sutures, and the patient was released on November 1.

The histologic examination of the excised lesion confirmed the clinical diagnosis of squamous cell carcinoma and established the tumor in grade II of Broders' classification. This patient has given up smoking tobacco completely, and has been seen at two month intervals for examination. When last seen, July 23, 1948, there was no evidence of recurrent cancer or enlarged cervical lymph nodes (Fig. 4, right). He will be examined indefinitely as a precautionary measure so that neck dissection can be undertaken promptly should metastatic lesions develop. This, however, is not expected due to the fact that the small primary lesion was excised early and widely.

■ *Cancer of the Gingiva*

Cancer of the alveolar ridge is of special interest to the dentist since patients with this neoplasm quite naturally believe that the disease is related to the teeth and seek dental consultation first. Martin[2] has pointed out that 59 per cent of a group of 100 patients with cancer

2. Martin, Hayes, Mouth Cancer and the Dentist. *J.A.D.A.* 33:845 (July) 1946.

of the gingiva first sought the advice of a dentist. This subgroup of 59 patients was studied further, and it was found that the dentist recognized the lesion as cancer in only 18 of the 59 patients, or 31 per cent of the group. The average delay between the first consultation with the dentist and consultation with a physician when cancer was suspected by the dentist was four weeks. In the 41 patients, or 69 per cent of the group in which the dentist failed to recognize the possibility of cancer, the average delay before the patient finally saw a physician was five months. This delay unquestionably lessened the chances of survival of this group.

EXTRACTION OF TEETH IN THE PRESENCE OF CANCER—The question of extraction of teeth in the presence of undiagnosed mouth lesions is also of interest to the dentist. If teeth are removed in the presence of undiagnosed cancer, valuable time will be lost waiting for healing before any attempt is made to diagnose the lesion. This delay may allow a cancer to progress to such an extent that early death is certain. The mistake of removing teeth in the presence of undiagnosed cancer is easily understood, for many patients make their own diagnoses, present themselves to the dentist and demand the extraction of teeth. These patients are displeased if the dentist refuses to extract the teeth to clear up an obscure gingival lesion, but should the tooth be removed and the diagnosis of cancer subsequently be made, these same patients may loudly blame the dentist. Therefore, the extraction of teeth in the presence of undiagnosed chronic gingival ulceration is dangerous to both dentist and patient.

Martin also studied this question of extraction of teeth in 122 patients with cancer of the mouth who saw a dentist first, and found that teeth were extracted in 70 per cent of the 72 patients in whose mouths cancer was not recognized. In the 50 patients in whom cancer was suspected, teeth were extracted in 30 per cent. A more detailed analysis revealed that in 29 patients with gingival cancer the dentist extracted teeth in 90 per cent of the patients when he failed to suspect the diagnosis of cancer. Extraction is unwise as it may allow invasion of the central portion of the alveolar process by the tumor, but even more important is the fact that extraction of teeth is always followed by a period of waiting to see whether cure of the lesion will result. The time thus lost before a diagnosis is made and proper treatment instituted cannot be regained.

PROGNOSIS OF CANCER OF THE GINGIVA—The history of cancer of the gingiva is a dismal one. Martin reports a 5 year survival rate of only 26 per cent of all patients treated. Only cancer of the tonsil, of all intraoral cancer, has a poorer prognosis. This fact may be due in large part to the failure of the dental profession to recognize early cancer.

REPORT OF A CASE—Figure 5, illustrates the only early cancer of the gingiva seen in the Oral Surgery Department in several years. This 74 year old white man (Fig. 5, left), was referred to the Oral Surgery Department of the Geisinger Memorial Hospital on January 27, 1948, complaining of a sore mouth of four months' duration. His present illness be-

Fig. 4.—Left: Squamous cell carcinoma of the right lateral margin of the tongue. Note depressed ulcer anterior to area of leukoplakia. Right: Right lateral margin of the tongue after wide excision of carcinoma

gan with a sort spot in the upper right molar region under his complete upper denture, which he had worn approximately five years. Two months after the onset of symptoms he consulted his dentist who took a smear of the lesion. The laboratory reported a moderate number of Fusiformis dentium and Borrelia vincenti. The dentist discussed the lesion with the pathologist and suggested immediate biopsy. The pathologist, however, advised a trial period of local therapy for Vincent's infection. Gentian violet was applied daily and the patient gave up smoking for a period of two months, but the lesion persisted and increased in size.

Examination revealed an ulcerative lesion approximately 1.5 cm. in diameter on the buccal surface of the alveolar process in the upper right molar region. The base of the ulcer was depressed and the margin was slightly elevated and rolled. The buccal tissue adjacent to the ulcer was indurated and adherent to the alveolar process. A white film covered the buccal mucosa and the palate.

On the date of his first examination a biopsy specimen was obtained using a 2 per cent procaine hydrochloride anesthesia, and blood was taken for a Wassermann test. He was referred to the medical department for a complete physical examination which revealed no contraindication to general anesthesia. The urinalysis was normal and an electrocardiogram revealed no evidence of myocardial damage. He was thought to be in excellent condition for a man 74 years of age. The Wassermann test was negative, but the histologic diagnosis of the biopsy specimen was squamous cell carcinoma (grade III according to Broders' classification).

SURGICAL TREATMENT FOR CANCER OF THE GINGIVA—Since this patient's general condition was excellent and the tumor was of relatively short duration and small size, an operation was planned to eradicate the tumor. He was admitted to the hospital on February 4, 1948, for operation the next day. He was given 1½ grains of propylmethylcarbinylallylbarbiturate ("Seconal sodium") one and a half hours before the operation, and ⅛ grain of morphine sulfate and 1/150 grain of atropine sulfate one hour before the operation. The larynx was anesthetized by topical application of a 10 per cent cocaine solution in order to avoid laryngospasm during passage of an endotracheal tube. At 1 p.m. a general anesthetic of a 2½ per cent solution of thiopental sodium was

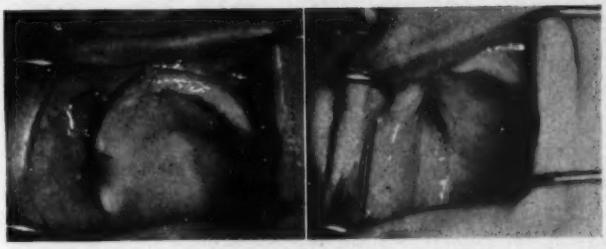

Fig. 5.—Left: Squamous cell carcinoma of the upper right edentulous alveolar ridge. Right: Oral fistula of right maxillary sinus after excision of carcinoma

administered intravenously. An endotracheal tube was passed through the nose and into the trachea. Anesthesia was thereafter maintained by administration of nitrous oxide and oxygen, supplemented as necessary by small amounts of thiopental sodium.

Using the electrocautery cutting current the tissue to be removed, which included a 1 cm. margin of normal tissue surrounding the tumor, was outlined. This electrocautery incision was carried through the periosteum of the maxilla and extended out into the cheek as far as the orifice of the parotid duct. This tissue was then elevated from the bone and removed from the mouth. The alveolar process and floor of the maxillary sinus were then removed with rongeurs. The entire wound was heavily cauterized with the electrocautery to destroy another centimeter of grossly normal soft tissue and bone. A 2 per cent gentian violet solution was applied to the wound, and the patient was returned to his room in good condition. During the procedure he received 500 cc. of 10 per cent glucose in normal saline solution intravenously. The patient was given 40,000 units of penicillin every three hours for four days to prevent infection. He was discharged on February 12, which was his seventh postoperative day.

On Feburary 16, he returned for observation complaining of restlessness. He was having considerable difficulty with speech due to the large opening into the maxillary sinus. Fluids also passed from his mouth to his nose adding to his apprehension. Therefore, he was given a prescription for phenobarbital, ¼ grain to be taken four times a day. He was seen for observation every two weeks until April 5, when a large sequestrum was removed from the maxilla without anesthesia. The wound healed quickly (Fig. 5, right), and by June 28, he was wearing new dentures made by the referring dentist. The upper denture closed the oral fistula of the maxillary sinus and restored nearly normal speech and function. When last seen on June 28, there was no evidence of local recurrence of the tumor or metastatic lesions in the cervical region.

■ *Conclusion*

The principles underlying management of cancer of the mouth are discussed and case histories are presented on cancer of the lip, buccal mucosa, tongue and alveolar ridge. The importance of early recognition of cancer is stressed.

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Some principles of bone pathology

■ *Harry Sicher, M.D., Chicago*

The literature on bone pathology is replete with controversial statements and classifications, and even the terminology is contradictory. This chapter of pathology is difficult, but laxity in the use of technical terms adds to the confusion. The persistence in the literature of hypotheses that never have been substantiated and the neglect of the wealth of evidence contradicting them, prolongs the period of uncertainty in the field. It is a truism that an understanding of disease can be founded only on an understanding of normal anatomy and physiology. This paper is an effort to clarify some aspects of the biology of normal bone that are of special significance to an understanding of problems of bone pathology and to indicate how some problems of bone pathology can be clarified by the application of the principles of bone biology.[1]

■ *The Elements of Bone Tissue*

Like other supporting tissue, bone tissue consists of living cells and intercellular substance. The cells, the osteocytes, by the fusion of their branching processes, form a syncytium throughout large areas of bone tissue. Thus the living elements represent a continuum, everywhere reducing to a minimum the thickness of intercellular substance through which tissue fluid has to diffuse. The intercellular substance itself consists of collagenous fibrils, arranged in small bundles, and a binding or cementing organic substance. Only the latter is impregnated with mineral salts, mainly calcium phosphates in the shape of submicroscopic hexagonal crystals of apatite.

Since the fibrils change their direction from layer to layer, normal mature bone appears lamellated (Fig. 1). Each lamella is from 4 to 12 microns thick.

■ *The Different Types of Bone Tissue*

With few exceptions, the bone tissue of the adult is of the lamellated type. However, the bone of the fetus, though it consists of the same elements, is characterized by a different arrangement of these elements. The cells of immature bone are much larger than the osteocytes of lamellated bone and lie more closely packed (Fig. 2). Thus in immature bone the amount of intercellular substance per unit volume is greatly reduced. Moreover the fibrils of immature bone are more numerous and coarser than those of mature bone. The intercellular substance in immature bone thus is relatively poor in cementing substance, and since only the cementing substance is calcifiable, the amount of mineral salts per unit volume

Read at the thirtieth annual meeting of the American Society of Oral Surgeons, Chicago, September 1948.

From the department of anatomy and research, School of Dentistry, Loyola University, Chicago.

1. Weinmann, J. P., and Sicher, Harry, *Bone and Bones*. St. Louis: C. V. Mosby Company, 1947.

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is much lower in immature bone tissue than in mature bone. The coarse fibrils of the immature, or coarse fibrillar, bone are not arranged in definite layers with a regularly alternating course, but are irregularly interlaced.

The replacement of immature bone by lamellated mature bone starts in the last weeks of intrauterine life, and continues through the first years, until all immature bone has disappeared.

Immature bone is characterized by its high radiolucency. This is, of course, related to the reduction of calcifiable cementing substance in a volume unit of coarse fibrillar bone because of the increased size and number of its cells and the increased caliber and number of its fibrils.

In many locations bone to which ligaments or tendons are attached also is of a distinct type. This kind of bone tissue is especially prominent in the alveolar process, where it has been described as bundle bone (Fig. 3). This term is derived from the fact that the bundles of the principal periodontal fibers continue as Sharpey fibers into the bone. The bundle bone is characterized by the scarcity of its fibrils, all of which are arranged in one direction, namely, at right angles to the Sharpey fibers. This uniform arrangement of the fibrils causes a lack of lamellation.

The scarcity of fibrils leads to a preponderance of calcified cementing substance in a unit volume of bundle bone, as compared with lamellated bone. The relative increase in cementing substance and thus in mineral salts per unit volume renders bundle bone more radiopaque than lamellated bone.

Many observations point to the probability that calcification of the cementing substance of bone (and dentin and cementum) is an all-or-none effect. In all probability there is no hypocalcification or hypercalcification of hard tissue. The varying mineral contents of these tissues seem to depend mainly on the ratio of the calcifiable cementing substance to cellular and fibrillar substances.

Fig. 1.—Decalcified section through human mandible in silver impregnation, showing alternating course of fibrils in adjacent lamellas of the matrix; X 1,500 reduced to about X 860. (From Weinmann and Sicher.[1])

■ *Formation and Resorption of Bone*

The questions of bone development and destruction seem to be the last battlefield of the defenders of humoral against cellular histogenesis. Followers of the humoral theory have ascribed to the osteo-

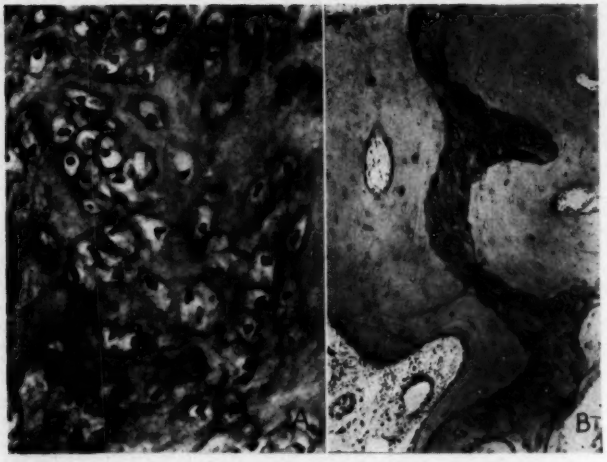

Fig. 2.—A: Decalcified section through the mandible of an infant, showing immature bone and irregular dense arrangement of large osteocytes; X 650, reduced to about X 298. B: Specimen from same section, showing replacement of immature coarse fibrillar bone by lamellated mature bone; X 180, reduced to X 82.5. The size, number and arrangement of osteocytes in the two types of bone tissue can be compared. (From Weinmann and Sicher.[1])

blasts and osteoclasts varying but secondary roles. According to these writers, for instance, the osteoblasts limit the production of bone tissue from the tissue fluids or from the connective tissue. Similar ideas of the role of odontoblasts in the pulp were suggested not long ago, and the process of bone destruction or resorption was called a "chemical" process without cellular activity. The proponents of these ideas have stated also that extraction of the mineral contents from the bone tissue during life not only is possible but that it is a most important step in bone resorption and that decalcification or demineralization may even be followed by remineralization or recalcification.

To the best of my knowledge, no proof for any of the foregoing hypotheses ever has been offered. Most of them are founded on the impossibility at the present time of setting forth the biochemical details of cellular bone formation or resorption. It is, however, apparent that the assumption of a humoral basis does nothing to lift the veil from these processes. Histologic pictures alleged to prove halisteresis, that is, decalcification of bone tissue, or fibrous degeneration of bone tissue are entirely unconvincing, partly because the histologic details manifestly are being misinterpreted. Tangential or oblique sections, for instance, sometimes give the illusion of a gradual transition of bone into fibrous connective tissue. Serial sections through such areas, however, reveal the true relations.

Faulty interpretation of roentgenograms and the continued use by radiologists of such terms as "decalcification" and "demineralization" have helped to keep alive the hypothesis of decalcification *in vivo*.

The evidence for cellular formation and resorption of bone, on the other hand, is overwhelming. Wherever and whenever new bone is formed, osteoblasts are lined up on the growing surface of the

bone tissue. When growth by apposition at a bone surface ceases, the osteoblasts disappear immediately. That is, they lose their typical appearance and arrangement and no longer can be differentiated from other cells of the connective tissue. It should be stressed, however, that osteoblasts are not always the perfect cuboidal cells the name suggests. They are often flat, sometimes even spindle shaped and not much different in shape from inactive connective tissue cells. Their relation to each other and to the bone and, most of all, the presence of a layer of uncalcified osteoid tissue between the cells and the surface of the calcified bone identify them as osteoblasts.

When growth at a surface of bone tissue has ceased and when the osteoblasts have disappeared, this surface may remain entirely inactive or aplastic for a long time. This is conclusive proof that the osteoblasts are not necessary for limiting production of bone tissue.

Though detailed biochemical knowledge of bone formation is still lacking, a tentative outline of the stages of this process can be given: The first product of osteoblastic activity is a seemingly homogenous organic substance consisting of, or differentiating into, fibrils and a cementing substance. The organic product of the osteoblasts is known as osteoid tissue. In a second phase this organic substance undergoes a decisive change, which can be compared to a denaturation of native proteins or to a progressive polymerization of proteins. Simultaneously mineral salts, mainly crystals of apatite, are precipitated in the cementing substance. Under normal conditions the formation of new bone is a rather slow process, and thus the osteoid layer often is extremely thin. Wherever bone development is accelerated, the "osteoid seam" is conspicuous.

It never has been shown that bone tissue can disappear in the absence of osteoclasts. Sections that show unmistakable signs of bone resorption but no multinucleated giant cells at the resorbed surface are understood easily if the rich experimental evidence on the life cycle of osteoclasts is not neglected. Their origin is from the cells of the loose connective tissue. In all probability they do not differentiate from capillary endothelium. Their differentiation occurs in a few hours

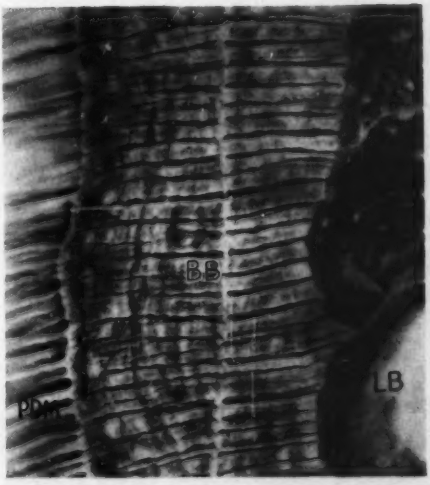

Fig. 3.—Alveolar bone in silver impregnation; X 330, reduced to about X 190. Bundles of principal fibers of the periodontal membrane enter the bundle bone as Sharpey fibers. Fibrils between Sharpey fibers are scarce in bundle bone, abundant in lamellated bone. PDM designates periodontal membrane; BB, bundle bone; LB, lamellated bone

after the proper stimulus has been applied. This stimulus can be, for instance, slight local pressure on a bony surface, as has been shown in orthodontic experiments, or the injection of parathyroid hormone. Whether the osteoclasts develop by fusion of uninuclear cells or by amitotic nuclear division is not clear yet, mainly because of the rapidity of their development. It has, however, to be stressed that an osteoclast is not always a giant cell with many nuclei. In Paget's disease, for instance, in which thousands on thousands of osteoclasts differentiate, uninuclear osteoclasts and, at the other extreme, real giant cells with up to one hundred nuclei occur. The eosinophil cytoplasm and its striations where the cell touches the bone are the most significant characteristics of the osteoclast.

There is ample experimental evidence that under normal circumstances osteoclasts survive only by a few hours their resorptive activity. Though under certain conditions, for instance in a giant cell epulis, the degeneration and dissolution of osteoclasts can be studied, nothing is known as yet about their dedifferentiation or degeneration in the normal tissues. Their disappearance is so fast that reliable observation of this phenomenon is extremely difficult. However, the observation of a bony surface immediately after osteoclasts had disappeared led to the unfounded assumption of humoral bone dissolution, osteolysis.

In weighing the evidence for osteoclastic bone resorption, one should remember that in orthodontic experiments and observations of large scale bone destruction after injection of parathyroid hormone, the histologic picture is dominated by the presence of many to innumerable osteoclasts. The study of clinical hyperparathyroidism, osteitis fibrosa generalisata, Recklinghausen's disease, or the study of active Paget's disease teaches the same lesson. The observations of Bloom and McLean[2] on the massive destruction of bone in the marrow cavities of pigeons during their egg-laying period spoke forcefully for the causal correlation of osteoclasts and bone resorption.

The problem of possible decalcification or demineralization of bone tissue *in vivo* (halisteresis of bone) is intimately linked to the problem of bone resorption. While there is not a shred of evidence for the reality of decalcification of bone *in vivo*, there is ample evidence that bone tissue disappears *in toto*, that is, that the organic and inorganic constituents of bone tissue are removed at the same time. Whenever uncalcified bone is seen in sections, it is possible to show that it is not decalcified bone tissue but uncalcified osteoid tissue. Incontrovertible evidence for the total and cellular destruction of bone tissue was reported by Bloom and McLean[2] from their study of the bone of egg-laying pigeons. Here calcium was stored as bone tissue, filling the marrow spaces of long bones. While this bone was broken down in a few days, innumerable osteoclasts could be seen at all surfaces of the bone tissue. Sections of undecalcified bone of these birds were examined by the von Kossa method for staining calcium salts *in situ*. Not the minutest area of bone tissue was found to be demineralized. All the bone tissue present contained normal and equal amounts of calcium. On the

2. Personal communication and lecture before the Chicago Institute of Medicine.

other hand, while the osteoclasts never contained calcium salts, the connective tissue of the marrow was filled with dustlike particles of calcium. Many macrophages were stuffed with the calcium salts.

From these findings and from many other less unequivocal observations, which, however, add an overwhelming sum of circumstantial evidence, one may draw the following conclusions: Bone tissue is destroyed only by the activity of specialized cells, the osteoclasts. Humoral osteolysis does not exist. The osteoclasts elaborate a proteolytic enzyme that digests or dissolves the organic constituents of bone tissue. The mineral salts are thus "liberated." If the resorption of bone proceeds slowly, as it does under normal circumstances, the calcium salts may go into solution in the richly flowing tissue fluid. If the destruction of bone is rapid, the mineral salts are taken up and transported away by macrophages. That osteoclasts speedily destroy bone tissue but are not able to destroy osteoid tissue is an added bit of circumstantial evidence for this interpretation of the osteoclastic activity. The organic parts of osteoid tissue change either by denaturation or by polymerization into those of calcified bone. Both processes are characterized by the fact that they lower the resistance of proteins to proteolytic enzymes.

It is time that authors who still cherish the ideas of humoral osteogenesis and osteolysis and the possibility or even the dominance of decalcification of bone *in vivo* take stock of the total sum of observations. The mere repetition of unsubstantiated theories will not replace factual evidence; it will only serve to perpetuate confusion and futile controversies.

■ *Bone and Bones*

It is of considerable importance to differentiate between bone as a tissue and a bone or bones as organs of the human body. Laxity in the use of the term "bone" has led to many erroneous concepts. It is well known that there are two types of bones, membrane bones that develop directly in the connective tissue of the embryo and endochondral bones replacing a cartilaginous model. Most textbooks speak of two types of bone development. This has been interpreted by many as indicating that the bone tissue of membrane bones is different in its biologic reactions from the bone tissue of endochondral bones. In reality, there is not the slightest histogenic, histologic or biologic difference in the bone tissue in the different bones of the body. Thus one should speak of two types of development of bones and realize that there is only one type of development of bone (tissue).

How important and illuminating this sharp differentiation between bone and bones is can best be made clear in a consideration of the growth of bones. A long or tubular bone grows thicker by the growth of bone tissue, but it grows longer by the proliferation of cartilage. That the growing epiphyseal and articulating cartilages, while they grow, are replaced by bone contributes not in the least to the elongation of the bone, but only to its consolidation. At the cranial base the overall growth occurs by growth of cartilage, but in the cranial vault and in the facial skeleton it occurs by growth of connective tissue in the sutures. Growth of bone tissue itself serves partly to thicken the bones of the skull and partly, by replacing the

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growing cartilage or connective tissue, to increase the solidity of the skull. Another point should be made unmistakably clear, namely, that bone tissue is not capable of expansive or interstitial, but only of additive or appositional growth. The apposition of bone tissue occurring inside a bone should not be confused with interstitial growth, because such apposition always is confined to free surfaces of the bone tissue that are in contact with connective tissue. It is clear that in this sense the surface of a spongy trabecula is a surface of bone tissue, though it is not part of the surface of a bone.

Finally, one should never forget that though bones are permanent organs of the body, bone tissue is, with few exceptions, a transitory element of the human skeleton. Throughout life bone tissue is in constant flux. Here and there overaged bone tissue is being resorbed, to be replaced by young, newly formed bone. During growth this process of internal reconstruction of bones is necessitated also by the steadily changing mechanical stresses on the skeleton.

BONE AND BONES IN THE ROENTGENOGRAM—Apart from revealing the shape of a bone and its structure, the relative density of the roentgenogram is an important clinical finding. However, it should be obvious that there are definite limits to the diagnostic value of a roentgenogram. The density of the shadow of bone is dependent only on the amount of mineral salts in the tissue. Since decalcification of bone tissue is not possible, a decrease of density means primarily a decrease in the volume of calcified bone tissue. Osteoid tissue as a rule is invisible in the roentgenogram. This means that osteoporosis and osteomalacia may give identical pictures. In both diseases there is loss of calcified bone; a simple loss without any regeneration in osteoporosis, loss of calcified bone and regenerative replacement with uncalcified osteoid tissue in osteomalacia.

However, loss of density can be caused also by loss of lamellated bone and its replacement with immature bone, which is much more radiolucent. Immature bone sometimes is so highly radiolucent that it cannot be detected in a roentgenogram. A defect in a bone may be filled with fluid, soft tissue or immature bone, without change in the appearance of the roentgenogram.

Bundle bone, on the other hand, is less radiolucent than lamellated bone and shows a heavy shadow in the roentgenogram. This has been interpreted wrongly as a sign of hypercalcification of bone tissue. Differences in the density of the lamina dura in the alveolar process find their explanation in the peculiar distribution of bundle bone. In a normal and complete dentition, bundle bone in considerable volume is found only at the distal side and at the fundus of the socket.

If the radiologist wants to avoid misinterpretation of his findings, he should not use terms like "decalcification," "demineralization" and "hypercalcification." More descriptive terms are most desirable. Also the term "swelling" should not be applied to a bone, because the actual condition always is an excessive growth of bone tissue.

DISTURBANCES OF GROWTH—Quantitative aberrations may affect bone tissue or bones. If the bone tissue is involved

(Fig. 4), its amount may be increased (osteosclerosis) or decreased (osteoporosis). Osteosclerosis may be caused by increased function, osteoporosis by a loss of function. The atrophy of disuse is an excellent example of the relation of structure and function. Especially in the jaws a series of atrophic stages can be observed (Figs. 5 and 6). The degree of osteoporosis parallels closely the progressive loss of function after extraction of one or several teeth in one or both jaws. The atrophy occurs not only at the site of extraction but in parts of the opposite jaw where teeth have lost their antagonists.

Osteoporosis as well as osteosclerosis involves both compact and spongy bone. In osteoporosis the campact laminas may disappear entirely if resorption starts at the outer surface, or may be replaced by spongy bone if resorption occurs at the inner surface. In the spongiosa each trabecula is thinned, while the marrow spaces widen. The exact opposite is, of course, true of osteosclerosis, where finally by thickening of all spongy trabeculas spongy bone may be transformed into compact bone.

Giantism and drawfism are, of course, growth aberrations of the skeleton as a whole. It is noteworthy that the extent of the ultimate change is dependent to a high degree on the involvement of cartilage and not only of bone tissue. Thus, for instance, an eosinophil tumor of the pituitary gland will cause true giantism in young persons who still possess epiphyseal cartilages in the tubular bones, while in older persons the growth will cause the picture of acromegaly. On the other hand, in chondrodystrophy, bone tissue is not at all involved. This type of dwarfism is caused by the suppression of the interstitial growth of cartilage.

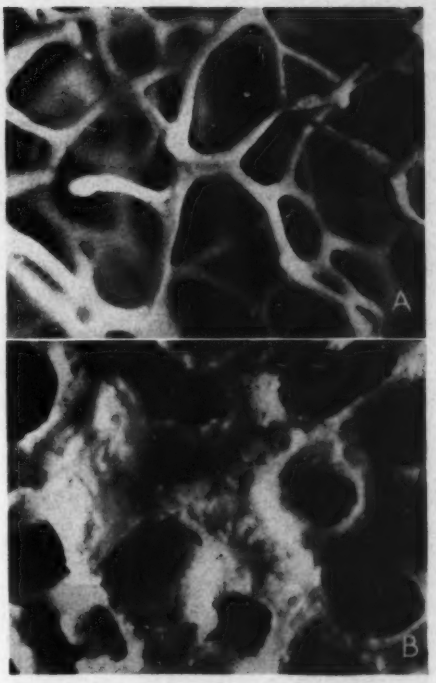

Fig. 4.—Magnified section showing, A, osteoporosis, and, B, osteosclerosis, of the spongiosa in a vertebra. (From Weinmann and Sicher,[1] after Putschar.)

DISTURBANCES OF OSTEOGENESIS—Bone tissue may suffer disturbances in its formation as well as in its calcification. For instance, the formation of the organic component of bone tissue is severely disturbed in vitamin C deficiency. In scurvy, the cells of the supporting tissues are not able to produce collagen, and the result is deterioration of connective tissues, cartilage and bone. Another formative defect of bone tissue is lack of proper organization

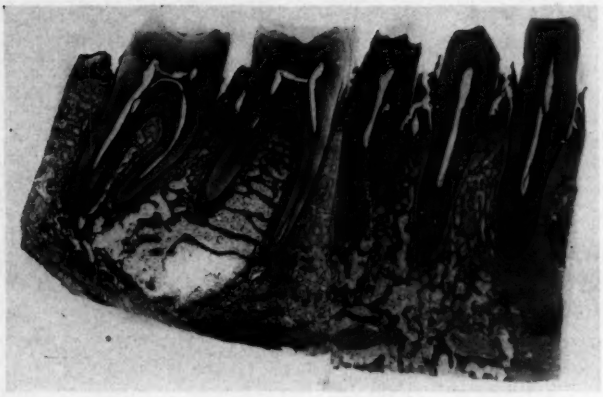

Fig. 5.—Sagittal section through the left mandible of a patient aged 42. (From Weinmann and Sicher.[1])

when immature bone instead of lamellated bone is formed, as in Recklinghausen's disease, in which under the influence of increased parathyroid function the lamellated bone gradually disappears and, in a futile attempt at regeneration, immature bone replaces it.

Defects of calcification, for instance in vitamin D deficiency, result in different clinical entities in children and in adults. The fast growing bones of infants suffer in rickets not only because of the persistence of osteoid tissue, but because of the inability of the growth cartilages to calcify. Lack of calcification prevents resorption not only of osteoid tissue but also resorption of cartilage and, therefore, the replacement of cartilage by bone. Thus in rickets the epiphyseal plates are thickened and irregular and finally cease to grow. The volume of osteoid tissue is greatly increased not as a compensation for its lack of mechanical strength but in consequence of a lack of modeling resorption. It is a fascinating and instructive task to analyze the rachitic symptoms and to recognize that the changes of the skeleton as a whole, that is, the changes of the bones, are caused by changes not only of bone tissue but also by the changes of cartilage.

In adults, deficiency of vitamin D or of calcium leads to the picture of osteomalacia, which means a softening of the bones and not a softening of bone tissue. The continuous reconstruction of bone tissue, that is, the resorption of old and its replacement by new bone tissue, is the key to an understanding of osteomalacia. The resorption of bone tissue continues at a normal rate or, in pregnant women, at an increased rate. Though bone tissue forms in fairly adequate volume, it never calcifies and persists as osteoid tissue. Slowly but surely the premorbid normal bone tissue is replaced by uncalcified osteoid tissue until the bones become soft and may then be deformed under the influence of normal function.

■ *Healing of Bones*

For an understanding of the principles of healing of fractures it seems necessary to review a few facts on clinical and experimental heterotopic formation of

bone tissue. From numerous observations one can conclude that proliferating connective tissue anywhere in the body may be the site of bone formation. It seems that especially the young and still relatively undifferentiated cells of loose connective tissue can be induced with various stimuli to differentiate into osteoblasts. One of the most general stimuli seems to be the presence of calcified or mineralized material. Thus, proliferating connective tissue in the neighborhood of bone is the most common source of the production of new bone. In this connection it should be remembered that a blood clot, if not infected, always is invaded by proliferating connective tissue and thus organized.

A question often discussed in connection with the healing of fractures is the osteogenic potency of the periosteum. The answers to this question have been and are contradictory. Apparently the periosteum as such, isolated from the bone it covers, is just a layer of connective tissue with all the general potentialities of such a structure. This explains the many experiments in which periosteum, transplanted into soft tissues of the body did not produce new bone. The opposite results, however, do not contradict this statement. Even in an adult bone, cells in the cambium layer of the periosteum may be activated from time to time—for instance, to replace resorbed bone tissue at the surface. Then in all probability the undifferentiated mesenchymal cells (Maximow) of the connective tissue are induced to differentiate into osteoblasts. At the time of an experiment the deep layer of the periosteum might contain cells that already had been stimulated by their closeness to bone and were already potential osteoblasts. Experimental embryologic studies have shown that cells once influenced toward a certain path of differentiation will follow that path even if they are removed

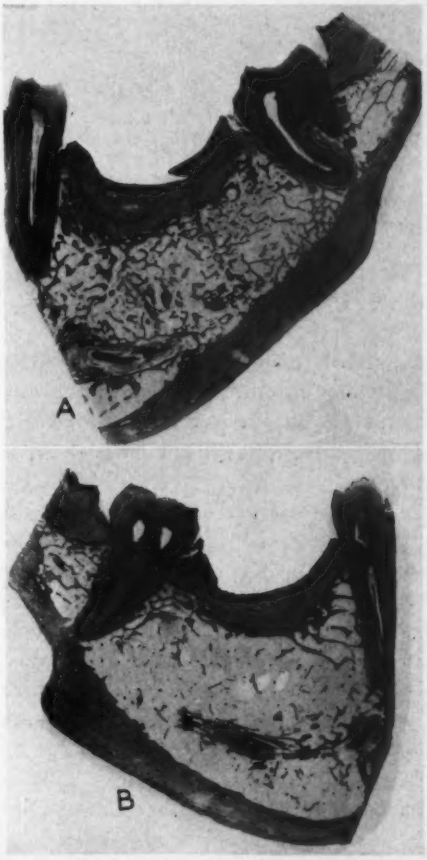

Fig. 6.—Sagittal section through, A, right side of lower jaw of patient aged 39, showing upper molars and moderate disuse atrophy; B, left side of lower jaw of same person, showing loss of upper molars and extreme disuse atrophy. (From Weinmann and Sicher.[1])

Fig. 7.—Socket of lower second bicuspid, three and one-half weeks after extraction; X 50, reduced to about X 27. The blood clot is organized, and the socket is half filled with rapidly forming immature bone. (From Weinmann and Sicher.[1])

from their original site. Potential osteoblasts may produce new bone, therefore, if the periosteum is transplanted. Once initiated, the process automatically continues.

The observation of excess formation of bone after a bone is stripped of part of its periosteum has led to the belief that the periosteum is a limiting membrane, keeping formation of bone tissue in check. It seems, however, that the overgrowth of bone tissue after removal of periosteum is caused not by the loss of this membrane, but by the secondary consequences of such an operation: formation of a blood clot, its organization by invading young connective tissue and finally the stimulation of the invading cells to differentiate into osteoblasts.

This interpretation of the functional properties of the periosteum has practical consequences. It is entirely a matter of chance whether implanted or carefully preserved periosteum will or will not produce new bone. The most important conditions for a favorable result are the presence of proliferating loose connective tissue and a stimulation of its cells to differentiate into osteoblasts. Implants of bone splinters or bone fragments or of artificial bone salts seem to act to maintain the proliferation of connective tissue and to provide osteogenic stimuli.

It has been stated repeatedly that there is some radical difference between the healing of fractures of mandible and maxilla and of fractures of other bones. Especially, the lack of callus formation, absence of cartilage in the callus and, in the upper jaw, the frequency of fibrous instead of bony union have been stressed. In this connection it has to be repeated that there is no structural or functional difference between the bone tissue in endochondral and in membrane bones. Differences in the mechanism of healing therefore cannot be explained by ontogenic differences of the involved bones. One has to remember, however, that practically all fractures of the jaws are compound fractures and as such will not heal like simple fractures. Secondly, in the upper jaw one has to differentiate between fractures that do not lead to functional mobility of the fragments and fractures that mobilize parts of or the entire upper jaw. That fractures of the first type heal not by bony but only by fibrous union is not surprising, because stimuli caused by

a certain degree of mobility seem to be essential to the regular formation of a callus, and especially to the formation of cartilage in the early stages of healing of bone. Crudely, fibrous union is comparable to a supernumerary suture.

The bone formed during the first stages of healing of a fracture or of a socket after tooth extraction is always immature, spongy bone (Fig. 7). The radiolucency of immature bone is the reason why a bony callus is invisible in the roentgenogram for a long time, and why a socket may appear empty though it is already filled with immature bone.

■ *Tumors of the Skeleton*

Any of the various tissues that make up a bone can give rise to a tumor. Such tumors can be classified as benign and malignant, the latter classification to be subdivided into primary and secondary tumors. As a special feature of tumors of the jaws, one could add tumors in the bones to tumors of the bones, in order to set apart the odontogenic tumors—for instance, the adamantinoma or ameloblastoma, the odontoma, cementoma and others. The table[1] summarizes the classification of tumors of the bones.

Only a few general facts about skeletal tumors can be discussed here. One of the most important is the pluripotentiality of young proliferating connective tissue cells. Especially if their mitotic rhythm is accelerated, they revert to a level of lower differentiation, though they can never be called embryonic cells. In a state of dedifferentiation (anaplasia) the connective tissue cells are sensitive to diversified

Tumors of the skeleton

TISSUE OF ORIGIN	BENIGN	MALIGNANT
Primary tumors		
Supporting tissues		
Connective tissue	Fibroma (ossifying) Myxoma	Fibrosarcoma
Cartilage Bone—osteoblasts	Chondroma (ossifying) Osteoma Osteochondroma	Osteogenic sarcoma
Accessory tissues		
Blood vessels	Angioma	Angiosarcoma
Adipose tissue	Lipoma (?)	Liposarcoma
Bone Marrow Blood-forming elements		Multiple myeloma
Reticulum		Ewing tumor
Secondary tumors		
Epithelium		Metastases of carcinoma
Mesoderm		Metastases of sarcoma
Tumors in the bone (odontogenic)		
Odontogenic epithelium	Ameloblastoma	Malignant ameloblastoma
Mesoderm	Cementoma	
Epithelium and mesoderm	Odontoma	

stimuli and may, after an otherwise subliminal stimulus, differentiate into fibroblasts, chondroblasts or osteoblasts. It is therefore erroneous to assume that the potentiality for cartilage formation is bound to cells of the endochondral bones and that displaced embryonic tissue elements are present if a chondroma develops in a membrane bone.

All the cells of the supporting skeletal tissues have the latent potentiality for producing bone, and this can become manifest in any one of the benign or maligant tumors arising from such tissues. That bone develops in fibromas or fibrosarcomas, in myxomas and in chondromas does therefore not need a specific explanation.

A second fact that has to be kept in mind, if one wishes to avoid unnecessary and misleading speculation and futile controversy, is the abnormal versatility of tumor cells. The tissues of tumors are often similar to, though not identical with, tissues of the normal body. This similarity can be utilized in labeling a tumor a myxoma, a chondroma or an osteosarcoma, but one should not go too far in such comparisons. It is, for instance, wrong to try to find all the elements of true mucoid connective tissue in a myxoma. It should be recognized that the cartilage of a chondroma can show structural details never seen in any of the normal types of normal cartilage. Especially the bone that is produced in benign or malignant osteogenic tumors can vary in its character so much that its identification as bone is almost arbitrary.

It seems arbitrary also to identify the specific type of blood cells in tumors of the hemopoietic marrow cells. Though they may resemble erythroblasts, plasma cells or myelocytes, they can have characteristics also that are alien to normal blood-forming cells. It is futile also to speculate on the origin of Ewing sarcoma from endothelial or reticular elements, because the sinusoids of the bone marrow are lined with reticular elements.

The reaction of bones to tumors finally merits some consideration. Tumors developing inside a bone will, of course, cause osteoclastic bone resorption in response to increasing pressure. However, it is important to note that resorptive activity is bound to the existence of a layer of connective tissue between tumor and bone. Once this layer has been destroyed and tumor cells are in contact with bone, resorption ceases. Splinters of bone can be seen embedded in tumor tissue, resembling sequestrums.

The progressing resorption of bone tissue on one surface and the consecutive weakening of the bone lead to a compensatory reinforcing osteoblastic activity on the other surface. The osteoblastic activity is in all probability the direct response to deformation of the weakened bone during function. The type of reinforcing bone formation depends largely on the speed of bone destruction, that is, on the speed of tumor growth. If the tumor grows slowly, then lamellated bone is apposed layer by layer, thickening the compact cortex or spongy trabeculas. If, however, bone destruction is rapid, then a peculiar framework of immature bones, the osteophytes, is produced. In this case one could speak of an emergency reinforcement. The osteophytes are short spicules of bone often arising at right angles to the original bony surface. After a short time they

mushroom out, developing trabeculas parallel to the surface of the old bone. From these fusing bars of bone a new row of osteophytes may grow, so that eventually several tiers of trabeculas are formed. As mentioned before, the osteophytic bone is always immature and always cancellous bone. These qualities are related to the speed with which the emergency reinforcement is built. Osteophytes are produced not only during the destructive growth of skeletal tumors, but as a response to other destructive bone diseases —for instance, osteomyelitis—or during intentional and therapeutic destruction of bone—for instance in orthodontic movement of teeth. The common denominator in all these circumstances is the rapid destruction and weakening of bone.

Certain metastatic bone tumors elicit a different and paradoxic reaction of bone tissue, namely, osteoblastic proliferation instead of osteoclastic destruction. Such tumors are metastases of carcinoma of the breast, bladder and prostate. Metastases from cancer of the breast elicit this reaction occasionally, those from prostate carcinoma almost always. This abnormal reaction of bone tissue has been attributed to the rich phosphatase content of the tumor cells. The lack of bone destruction and the formation of new bone tissue on the walls of invaded marrow spaces lead to a creeping growth of such metastases, which spread through the marrow spaces and haversian canals. These metastatic bone tumors, therefore, often defy radiologic detection.

Preoperative treatment and study of carcinoma patients

■ *Frederic W. Coggan, D.D.S., Kalamazoo, Mich.*

The necessity for cooperation between dentist and physician cannot be overstressed in the treatment of oral cancer. If a differential diagnosis has revealed the patient to have complications of tuberculosis or syphilis, an effort must be made to restore the patient to health, and to avoid procrastination in the treatment of cancerous lesions of the oral cavity. Heart disease, diabetes, anemia and other general systemic disorders must likewise be recognized and treatment instituted if a pleasant result is desired. In order to determine that a lesion is a primary malignant oral lesion that perhaps has not metastasized, a differential diagnosis between cancer, tuberculosis and syphilis must be carried out. Roentgenograms of the chest, skull, spinal column and pelvis will rule out the possibility of distant metastases.

Read at the annual clinic day of Oral Surgery Department of the Henry Ford Hospital.

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■ *Physical Examination*

The majority of cases of oral cancer are in the older age groups and an evaluation of the patient's physical condition is essential. Dentists should certainly stress the necessity of strict oral hygiene. Any evidence of Vincent's infection should be eliminated. If gross deposits of calculus are present, a dental prophylaxis is essential. Irritating cusps, tooth remnants or snags, ill-fitting dentures, poorly adapted clasps and obvious dental foci should be eliminated. If irradiation of the lesion is to be done, any teeth in the direct path of treatment must be removed before such therapy is started.

As it is essential to have all patients hospitalized, the first evaluation of the patient's general health will come from a thorough physical examination. If normal, other consultations may not be necessary. However, many patients, being of advanced age and perhaps malnourished, might need dietary and vitamin therapy. A few days spent in supportive treatment may save many anxious moments in both postoperative complications and other complications not related to the serious oral disease. It is obvious that the hazard of operating on or treating these patients may increase or decrease with either a high or a low index of general health.

The time element involved, measured from the first knowledge the patient has of something wrong until he comes for diagnosis, is a factor bearing directly on his physical status before treatment is undertaken. He may have delayed for weeks or even months, allowing physical debilitation and general body damage from effects of the progress of his oral disease or his inability to masticate and ingest food properly. Therefore, this patient can be expected to be a victim of poor nutrition, particularly if he has been slow to seek help. This patient must have a scientific investigation of his general physical condition, and the proper measures must be instituted to correct certain abnormal conditions before any attempt at surgical treatment is considered.

The general medical consultant must give his evaluation of the condition of the patient's heart and the possible existence of any pulmonary disease. Chronic diseases such as diabetes, or rare diseases such as the leukemias must also be ruled out.

■ *Laboratory Tests*

Laboratory tests are expensive but there are several that may have to be done in order to arrive at a proper evaluation. Frequent procedures are (1) total blood protein, (2) ascorbic acid level, (3) hemoglobin, red and white cell counts, (4) blood urea nitrogen, (5) bleeding and clotting time, (6) blood prothrombin, (7) blood Kahn and (8) urinalysis.

The total blood protein is normally 6.5 to 8 Gm. per hundred cubic centimeters and is usually subdivided into albumin and globulin. A normal ratio is albumin 4.5 to 6.5 Gm. per hundred cubic centimeters to globulin 1.5 to 2.5 Gm. per hundred cubic centimeters. When the blood protein level is below normal, fluid leaves the blood vessels and the result is edema. Edema does not necessarily impair healing to a great extent, but it is a warning that the nutritional state of the patient is not good.[1] Concentrations of

1. Nash, Joseph, *Surgical Physiology*. Springfield: Charles C Thomas, 1942.

blood protein below 6 Gm. per hundred cubic centimeters may signify a moderate grade of nutritional deficiency. Rather than wait to increase the blood protein by dietary control, amino acids given in the form of "Amigen"[2] will usually restore the percentage to a desired level.

If a hypoproteinemia exists and it is thought advisable to defer surgical intervention for a few days, the patient can usually be reverted from a chronic negative to a positive protein balance by diet. When the patient is in balance, the intake of protein is equal to the loss. The loss of protein is measured by the amount of nitrogen in the urine multiplied by 6.25. Hypoproteinemia retards all healing processes, whereas a high protein intake favors wound repairs.[3] The soft and liquid Varco[4] diets and modifications give adequate nourishment in either small or variable quantities. An intake of 80 Gm. of protein should be the minimal dietary requirement, but some patients might be favored by a daily intake of 120 Gm.

The ascorbic acid level is normally about 0.9 to 1.5 Mg. per hundred cubic centimeters of blood. A deficiency of vitamin C slows or stops the healing or reparative processes. Ascorbic acid is thought to be a detoxifying agent of the body. The vitamin C content of the blood is an accurate index of the nutritional status of the patient, and this level can easily be improved if necessary by the daily intravenous injection of 500 Mg. of ascorbic acid. It might also be wise to give daily doses of 300 to 450 Mg. of ascorbic acid orally. Because citrous fruits, the rich food source of vitamin C, cause considerable pain to raw oral lesions, it is not surprising to find low levels of ascorbic acid in individuals with malignant lesions of the oral cavity.

The blood urea nitrogen is normally about 14 Mg. per hundred cubic centimeters of blood. If a kidney insufficiency is present, excretion of this end product of metabolism is diminished. Hence, a high urea nitrogen content in the blood is indicative of poor renal function, or perhaps dehydration.

Hemoglobin determination and red and white cell counts are excellent indicators of the patient's general health. The white cell count is normally 7,000 per cubic millimeter of blood; noticeable variations may indicate a leukemia or agranulocytic angina. The normal red cell count varies between 4,500,000 per cubic millimeter in females and 5,500,000 per cubic millimeter in males. A large decline in red blood cells results in anemia. One hundred per cent is considered as normal hemoglobin content of the blood, or it may sometimes be classified as 15.6 Gm. of hemoglobin per hundred cubic centimeters of blood.

Since the hemoglobin transports the oxygen to the cells and tissues of the body and thus determines their vitality, any abnormal findings should be corrected if possible. If the hemoglobin is below 75 per cent, the patient should receive a pint of whole blood before surgical intervention. If considerable surgical blood loss is anticipated, the patient should be fortified preoperatively with

2. "Amigen" (Mead Johnson) given intravenously contains 100 Gm. protein in 1,000 cc. of 5 per cent dextrose.

3. Harding, Robert L., The Use of Protein in Oral and Maxillofacial Surgery. *J. Oral Surg.* 6:235 (July) 1948.

4. Varco, R. L., Preoperative Dietary Management for Surgical Patients, with Special Reference to Lesions of the Stomach and Duodenum. *Surgery* 19:303 (March) 1946.

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transfusions. Transfusions also supply substances responsible for normal coagulation and are of great benefit in various hemorrhagic diseases.

Bleeding time and clotting time are important and are often used to differentiate hemophilia and purpura. Normally, bleeding time is from 1 to 3 minutes, and clotting or coagulation time from 3 to 6 minutes. Purpura is characterized by a low platelet count (perhaps as low as 10,000 per cubic millimeter instead of 200,000 to 300,000 per cubic millimeter). The clotting time is normal but the clot does not retract and it is soft. The bleeding time is prolonged. In hemophilia, the bleeding time is usually normal, but the clotting time is prolonged.[5]

The blood prothrombin level is normally 100 per cent; or if spoken of as prothrombin time, the normal is 12 seconds. Prothrombin is presumably formed in the liver from a fat soluble substance, vitamin K.[1] In the normal clotting of blood, prothrombin in the presence of calcium produces thrombin. Fibrinogen is then activated by the thrombin to form fibrin which forms the network of the clot.

A low prothrombin level may be improved easily and rapidly by the administration of a vitamin K analogue (menadione sodium bisulfite) given hypodermically (one, or more, 5 Mg. ampul per day). This eliminates the necessity of inexact oral adminstration and the undesired effect of the use of bile salts with the vitamin.

The urinalysis is an important laboratory test used mainly for the detection of sugar or the diagnosis of diabetes. Albumin discovered in the urine is usually a sign of renal damage. If white blood cells are present, a genito-urinary infection is indicated. The presence of red blood cells may indicate a tumor, stones, or tuberculosis in the genito-urinary tract.

The blood Kahn is of great importance in the differential diagnosis. However, a chancre may resemble a carcinoma, and the Kahn may be negative in this early stage of syphilis. It is also possible for a carcinoma and a gumma to coexist. A dark field examination and a biopsy may both be necessary to arrive at a correct preoperative diagnosis.

Before the operation the patient must have an adequate fluid intake. If the daily urine output is less than 1,000 to 1,500 cc., the operation should be delayed until a forced fluid intake gives an output in the desired bracket.—*804 American National Bank Building.*

5. Macleod, J. J. R., *Physiology in Modern Medicine*, ed. 8. St. Louis: C. V. Mosby Company, 1938.

■ Eleventh Congress of ARPA Internationale

The Eleventh Congress of ARPA Internationale will be held at Groningen (Netherlands) June 28 to July 2, 1949. Subjects to be discussed include (1) nomenclature and classification; (2) functional treatment; (3) orthodontics and periodontal diseases.

The Eleventh Congress, organized by the Dutch Section of ARPA under the direction of Dr. Duijvensz, will be presided over by Dr. J. C. R. Jaccard, president of ARPA Internationale. For further information, write to Dr. Michaelis, Oilhilminapark, 5-Oegsteest, Netherlands.

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Postoperative care following carcinoma surgery

■ *L. W. Peterson, D.D.S., St. Louis*

The postoperative care and treatment of the patient who has submitted to radical procedures for removal of malignant carcinomatous lesions are often of equal importance to the operation performed as far as the patient's chance for recovery is concerned.

The immediate postoperative care begins with the orders left by the surgeon for the welfare of his patient. It then becomes the responsibility of the resident and nursing staff to carry out these directions. The orders follow a general pattern and include (1) maintenance of an adequate airway, (2) control of hemorrhage, (3) diet and fluid balance and (4) sedation.

Pertinent points relative to these groupings will be discussed in the order given. Further consideration will be given to dressings, wound healing and prevention of complications.

■ *Maintenance of Airway*

In all cases of operations about the mouth, the patient is placed on his abdomen or chest so that fluid can escape out the mouth. Suction equipment is kept at the bedside and the patient is never left alone until fully reacted. Oxygen-carbon dioxide inhalations are used to shorten recovery time and minimize possible pulmonary complications. The inhalations are given every 15 or 20 minutes, or at the time blood pressures are checked. The inhalations are a mixture of 70 per cent oxygen and 30 per cent carbon dioxide.

When a mandible is partially or totally resected, stabilization of the jaw through intermaxillary fixation, internal wire or bar splinting (Fig. 1), aids in supporting the tongue and maintaining the position of the mandible.[1] This wire will also stabilize the two bodies of the mandible when the symphysis is removed. Elastics are preferred for intermaxillary fixation until danger of nausea and vomiting is past. Tongue sutures for traction can be left in place until the patient reacts.

Patients with actual or suspected respiratory difficulty are watched closely. Signs of upper airway blockage are (1) unexplained and increasing restlessness, (2) intercostal and suprasternal retraction with each inhalation, with occasional dilatation of the alae nasi, (3) increased pulse rate which means that the heart is being taxed and (4) cyanosis, a late sign

Read at the annual clinic day of the Oral Surgery Department of the Henry Ford Hospital.

This paper is based on the functioning of the Maxillofacial and Plastic Surgery Division of Barnes Hospital, St. Louis, Missouri. The writer wishes to acknowledge the cooperation of Gordon Letterman, M.D., resident in plastic surgery.

From the Department of Oral Surgery, Washington University School of Dentistry and the Department of Surgery, Washington University School of Medicine.

1. Byars, L. T., Subperiosteal Mandibular Resection with Internal Bar Fixation. *Plast. & Reconstruct. Surg.* 1:236 (Sept.) 1946.

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which is usually followed by a drop in blood pressure and loss of consciousness.

Tracheotomy should be done before cyanosis occurs unless other measures such as adjustment of the airway, suctioning the throat, or loosening bandages give relief.

When necessary, tracheotomy should be performed early, and in the operating room if possible. It is often a wise precaution to keep a tracheotomy set, procaine hydrochloride and gloves, ready at the bedside. Anyone doing extensive surgery about the head and neck should be thoroughly familiar with the tracheotomy routine which will not be discussed in detail here.

The trachea should be opened below the first tracheal ring to prevent a possible laryngeal stenosis. Care of the tracheotomized patient is also important. A feeding tube should be used if the patient swallows with difficulty, and too much liquid should not be given at any one time to avoid regurgitation and overflow into the larynx. Moist inhalations and the use of ammonium chloride or potassium iodide may be advantageous if secretions are thick. Both inner and outer tubes must be kept scrupulously clean. The patient should not try to talk, but have paper and pencil handy for all communications. Care must be taken in handling the tracheotomy tubes to avoid irritation of the trachea.

■ *Hemorrhage*

A complaint of bleeding whether made by the patient, nurse or relative should always be investigated. Primary hemorrhage is usually well controlled before the patient leaves the operating room. The use of absorbable sponges has minimized secondary bleeding. Pressure dressings and obliteration of either soft tissue or bone cavities by packing are also invaluable in controlling hemorrhage. Most bleeding about the face and jaws can be controlled by pressure if properly and adequately applied. Sterilized surgical waste makes a good pressure dressing when used in bulk under a gauze roll and gauze bandage head dressing (Fig. 2).

Preoperative ligation of large vessels is not satisfactory in the control of bleeding about the face because of the extensive collateral circulation. Hemorrhage is best controlled through local efforts at the site of bleeding. Wounds coming in contact with each other, or having their dirty surfaces bathed in their own secretions, mean almost certain secondary hemorrhage. At all times these regions must be well dressed and bandaged.

Fig. 1.—Left: Posteroanterior postoperative roentgenogram showing internal wire splint in place. Right: Illustrating function and position of mandible with wire splint in position

■ *Shock*

Harkins[2] considers shock to be initiated by traumatic local fluid loss, either whole blood or plasma. Many other factors such as an associated vasoconstriction, decreased cardiac output and a decreased blood flow leading to anoxia and capillary wall and cell injury accompany and perpetuate the condition. The etiology of traumatic and hemorrhagic shock is basically the same.

The symptoms of shock are very similar to those of upper airway blockage. These include: an ashy gray color; cold, clammy skin; fast but weak and irregular pulse; progressive fall of blood pressure; irregular, inefficient breathing. Although blood pressures are checked frequently until stable, a fall in blood pressure is an inadequate guide to the state of circulation in incipient shock. In secondary shock a fall in blood pressure is often a terminal phenomenon occurring long after hemoconcentration and decreased blood flow have become evident. However, a progressive fall in blood pressure might indicate continued blood or plasma loss from the circulatory system with the shock condition inevitably ensuing.

There are two aims in the treatment of shock. These are replacement of fluid loss and prevention of further loss of fluid. Whole blood or plasma can be used to replace the loss of fluid. In advanced shock which tends to become irreversible even after replacement of more fluid than is actually lost, the two best remedies at present seem to be oxygen and adrenal cortex extract. The oxygen prevents anoxia and the adrenal extracts prevents increased capillary permeability.

Fig. 2.—Gauze roll and gauze bandage head dressing over sterilized surgical waste

■ *Diet*

The value of proper nutrition in the recovery of patients with various diseases is so important that the surgeon has been forced to be more concerned with proper diet and diet supplements. Postoperative diets are given by oral intake, tube feedings or intravenously as the occasion demands. Tube feeding is preferred to the intravenous method if the patient is unable to swallow. Actual blood loss is usually replaced at the time of the operation, so the postoperative fluid balance problem is largely concerned with water replacement, with chlorides and glucose added as necessary. A calculated amount of liquid must be given to allow for fluid loss through vaporization and urine output along with the body's other nutritional requirements.

Glucose serves two purposes; it pre-

2. Harkins, H. N., Recent Advances in Study and Management of Traumatic Shock. *Surgery* 9:231 (Feb.) 1941; 9:447 (March) 1941; 9:607 (April) 1941.

vents starvation ketosis and aids in protein metabolism. There are three factors that contribute to immediate postoperative hepatic glycogen deficiency. These are: glycogenolytic effect of certain anesthetics, inadequacy of oral food intake and the disease itself.

Pareira and Somogyi[3] found that an average of 200 Gm. of glucose per 24 hour period provided enough glycogen to the liver to prevent ketosis. They also used 10 per cent glucose in intravenous feedings and found no contraindications to its use when infused at the proper rate.

Maddock and Coller[4] concluded that 3,500 cc. of fluid is the minimal daily intake to provide for normal body functions. This allows for a 2,000 cc. loss through vaporization and a 1,500 cc. loss through the urine, which is the minimum daily output to take care of excretion of waste material by the kidneys in all ranges of function. Abnormal losses due to excessive vomiting, drainage from wounds or perspiring must also be replaced. An extra volume is necessary when dealing with a dehydrated patient. Patients are given fluids parenterally because of inability to take orally an amount sufficient for normal balance.

Murphy[5] warns against the indiscriminate use of intravenous fluids especially for patients with cardiovascular defects. Certain safeguards must be adhered to strictly. Fluids should be given slowly, in an isotonic solution, and in small amounts. Fantus[6] states that the postoperative use of fluids, to be properly individualized, demands observation of balance between fluid intake and fluid elimination, determining the percentage of chloride in the urine and the testing of it for sugar.

Number 14 or 16 soft rubber Levine stomach tubes are placed when a patient is unable to swallow, and are commonly used after neck dissection. The tubes are inserted either at the time of the operation or when nausea occurs. When a stomach tube is placed it should be tested immediately to see that it is not in the trachea. A small amount of fluid should be injected after aspiration tests and air sounds

3. Pareira, Martin, and Somogyi, Michael, Rationale of Parenteral Glucose Feeding in the Postoperative State. *Ann. Surg.* 127:417 (March) 1948.

4. Maddock, W. G., and Coller, F. A., Water Balance in Surgery. *J.A.M.A.* 108:1 (Jan.) 1937.

5. Murphy, F. D., Correll, Howard, and Grill, J. C., The Effects of Extravenous Solutions on Patients, With and Without Cardiovascular Defects. *J.A.M.A.* 116:104 (Jan.) 1941.

6. Fantus, Bernard, Fluid Postoperatively; A Statistical Study. *J.A.M.A.* 107:14 (July) 1936.

Table 1

Breakdown of 2,500 calories recommended for a 154 lb. (70 Kg.) man for a 24 hour period

Average feedings	Grams	Calories
Protein	125	500
Fat	100	900
Carbohydrate	300	1,200
Total	525	2,600
Average tube feedings	**Grams**	**Calories**
Protein	75	300
Fat	100	900
Carbohydrate	350	1,400
Total	525	2,600

Table 2

Diet suitable for all cases requiring liquid feedings

Liquid feedings	Ounces	Centimeters	Protein grams	Fat grams	Carbohydrate grams	Calories grams
6 eggs	6	180	36	30		450
20% cream	8	240	8	48	8	480
Milk	24	720	24	24	48	480
Tomatoes (strained or canned)	6	180	3		6	36
Glucose (sugar, Karo)	10	300			300	1,200
Salt (1 tsp.)						
Total	54	1,620	71	102	362	2,646

indicate the tube is in the stomach. If there is no cough or distress from 4 to 6 cc. of sterile water, the tube is probably in the esophagus or stomach. The tube should then be adjusted so that the injected contents can be completely aspirated back into the syringe. A golf tee or rubber band should be used to close the tube to prevent regurgitation when the patient coughs or moves around in the bed.

A breakdown of daily calory intake, as recommended by the Food and Nutrition Board, National Research Council, is shown in Table 1.

A liquid diet that can be used in all cases requiring feeding tubes, or in cases where a liquid diet is necessary, is shown in Table 2.

These ingredients are mixed together and divided into five feedings, given slightly warmed and at a swallowing rate. When possible, the liquids can be given unmixed and served as appetizingly as possible. Fruit juices, water, coffee, tea and ice cream can be given between feedings. Fluid from ground lean beef soaked in water may be given for additional protein. Six ounces of "Dryco" or dextrin can be substituted if the volume is too great for a 24 hour period.

Unless the operation presents some contraindications, most patients can be put on a regular diet the day after the operation as far as the general anesthetic is concerned. Patients who have no teeth or dentures, or who have jaws immobilized, should have a nonchewing diet, which is nothing more than a regular diet put through a food grinder or puree mill. Special diets may be ordered on occasion, but they must be appetizing. Dietitians are available in most hospitals to help if a patient is not eating.

■ *Sedatives*

The usual postoperative order for pain is 1 grain of codeine combined with 10 grains of aspirin given every 4 hours if necessary, and 1½ grains of pentobarbital sodium as necessary for sedation. Codeine may be given hypodermically if the patient is nauseated, or morphine may be substituted if it is thought the patient will have an unusual amount of pain, or if he has a low threshold to pain. Morphine should not be given to a patient with questionable respiratory embarrassment or depression. Infants and very young children should have little or no postoperative sedation unless they are unusually restless. Older children may have

small doses of aspirin or codeine when absolutely necessary, and 4 cc. of "Elixir of Luminal" at bedtime if needed. Opiates are dangerous to children unless carefully controlled. Patients should be checked regularly to see if they are being kept fairly comfortable. Writing p.r.n. orders does not always suffice, as patients may not know how or when to ask for the sedation.

■ *Dressings and Bandages*

Tight bandages can ruin a postoperative course. All complaints should be investigated carefully, not merely resorting to sedatives or anodynes to give relief. Eye bandages should be removed when complained of, as corneal ulcers and conjunctivitis from ether or other chemicals or from foreign bodies are very serious complications and every precaution should be taken to prevent them. Use cotton with a solution of saline, boric acid, or a 1:5,000 aqueous solution of "Zephiran chloride" for eye sponges. Benzin is very useful for removing adhesive tape with minimal pain and there is seldom any excuse for removing tape without it. Ether is not as good and it nauseates patients who have had a general anesthetic. Alcohol frequently causes a burning sensation when applied to skin that has been covered with adhesive, but if it is put over small areas at a time and washed off immediately with water or boric solution, less pain will result and a large field can be cleaned.

In wound healing simple mechanical cleanliness is of primary importance. Open wounds are always dressed with fine mesh gauze either wet or greased. Wet dressings must have plenty of bulk and waterproof coverings, so they will stay wet, or have rubber tubes incorporated in them for frequent addition of fluid. Greased dressings consist of one layer of fine mesh gauze greased with any bland ointment, with all excess grease scraped off, and usually covered with a large dressing. These are used only on clean wounds and are to be changed every one or two days as necessary. Grease crusts around the margin of the wound should be removed with ether or benzin and the whole wound washed gently with white soap and water. When sutures are placed they are carefully removed as soon as one is assured that the wound will not separate. The wound edges are supported for a time with fine mesh coat in gauze adhesive, elastoplast or some combination of these.

HEALING—Wounds heal spontaneously by contraction of surrounding edges and by filling in with fibrous tissue, and by scar epithelium going across from side to side. Skin is a complex organ and epithelium is the only part of it that regenerates. Rapid healing usually occurs in the face even after full thickness losses, presumably from deep hair follicles that extend into subcutaneous tissues (in men). Deep fibrous healing is presumably the only mechanism in which defects below skin level finally close, the region granulating in and gradually changing to fibrous tissue.[7]

Pressure dressings are of fundamental importance for good quick healing and are used on all wounds unless there is

7. Brown, J. B., and McDowell, Frank, Epithelial Healing and Transplantation of Skin. *Ann. Surg.* 115:1166 (June) 1942.

some special reason for omitting them.

Surgical defects in the oral cavity are usually packed with iodoform gauze saturated in Balsam of Peru. The Balsam of Peru masks the fetid odor accompanying oral dressings. These dressings are not removed until the sixth or seventh postoperative day. The dressings are then changed at three to five day intervals as healing progresses, and until the wound is granulating in satisfactorily. When cautery is used on bone, it frequently requires a long time for necrosis and sequestration, and it is not necessary to continue dressing the wound once the edges show contraction and begin filling in with fibrous tissue.

After neck dissections, pressure dressings applied at the time of the operation should be changed in 48 hours. Drains are left in place 7 days. Flaps should be held down as well as possible to keep fluid from collecting under them. Sutures can be removed in 7 to 10 days if flaps are healed well enough.

INFECTION—The possibility of secondary infection is minimized by mechanical cleansing with mouth washes and the use of 1 per cent gentian violet, 1 per cent acriflavine or in combination. Proper and adequate oral hygiene is an essential counterpart to a postoperative course following tumor surgery in the mouth. Chemotherapy also is an adjunct to a smooth, uncomplicated postoperative course.

RADIATION THERAPY—Therapeutic radiation will produce a radio-epithelitis on the thirteenth or fourteenth postradiation day. This may resemble stomatitis or Vincent's infection, and in the pharynx it is sometimes confused with diphtheria. Radiotherapy will often lead to necrosis of bone and an osteomyelitis which must be controlled. Preoperative extraction of teeth, regardless of their condition, in an involved region is an accepted procedure.

Often there are two factors a patient must contend with following radiation therapy. These are excess salivation and trismus. This period of excess salivation is more limited when interstitial radiation is used. The patient should be kept well nourished and have plenty of fluids. Sticky or mucoid salivation can be helped by giving potassium iodide for a few days.

The trismus which follows radiation and surgery about the head and neck may be due to the lesion itself, the mechanics of its removal, or it may be a protective mechanism. Efforts should be made to maintain good oral hygiene and mild jaw exercises should be instituted.

■ *Postoperative Complications*

Most postoperative complications can be avoided or minimized by attention to details and proper nursing care.

Pulmonary involvements are avoided by postural drainage, suction removal of secretions, frequent changes of position and having the patient sit up and walk as soon as possible. Antibiotics are also used.

Thrombosis, or embolism, is relatively rare after operations about the head and neck, but gentleness in handling tissues, prevention of dehydration and shock and frequent shifting of position are acceptable precautionary measures. Anticoagulants are employed at the first indication of thrombosis. Chemotherapy

also is used when treating a thrombophlebitis.

It is vital to maintain proper elimination of body waste at all times, using laxatives and enemas as indicated and catheterizing if necessary. A patient who is vomiting for no apparent reason might possibly have a fecal impaction or be sensitive to opiates.

Parotitis after oral operations is an infrequent complication. Proper preoperative oral hygiene is the best preventive. When parotitis does occur, frequent stimulation with lemon juice or chewing gum, and gentle probing of the duct may aid in the treatment. Pilocarpine, 1/120 grain every 4 hours for adults, external heat and roentgen therapy are other adjuncts. Chemotherapy is a preventive and also an effective treatment. This type of parotitis rarely needs external drainage.

■ *Conclusion*

The coverage of postoperative care by not being individualized is by no means complete. In carcinoma surgery, one deals primarily with older patients who have the usual associated debilitating diseases and infirmities. In this era of specialization, the valued consultation of a good internist will frequently aid in treating the patient who has a complicated medical history. With the ever increasing knowledge of medical and surgical treatment, no one man can hope to carry the complete responsibility if the best interests of the people are to be served.

■ Seminar for the Study and Practice of Dental Medicine

The sixth annual Seminar for the Study and Practice of Dental Medicine will be held October 23 to 28, 1949, at the Desert Inn, Palm Springs, California. The dates are conveniently arranged to enable those persons interested to attend both the Seminar and the American Dental Association convention, which will be held October 17 to 21, in San Francisco.

Applications are currently being accepted, and inasmuch as facilities are limited, early arrangements should be made by contacting Miss Marion G. Lewis, Executive Secretary, 1618 Ninth Avenue, San Francisco 22, California.

Seven outstanding speakers will discuss and compare the most recent discoveries in dentistry and the related fields of medicine and biology. They include: Arthur C. Curtis, M.D., professor and director of the Department of Dermatology and Syphilology at the University of Michigan; Wilton Marion Krogman, Ph.B., M.A., Ph.D., professor of physical anthropology, University of Pennsylvania; Seymour M. Farber, M.D., assistant clinical professor of medicine, University of California Medical School; Balint Orban, M.D., D.D.S., professor, University of Illinois; D. Harold Copp, M.D., who is collaborating with the Division of Dental Medicine, University of California; Francis A. Arnold, Jr., B.S., D.D.S., senior dental surgeon, U. S. Public Health Service; Paul Popenoe, Sc.D., director of the American Institute of Family Relations, Los Angeles.

President of the Seminar is Dr. Hermann Becks, chairman of the Division of Dental Medicine, College of Dentistry, University of California.

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Hemorrhagic conditions of importance in oral surgery

■ *O. Lee Ricker, D.D.S., M.S., Grand Rapids, Mich.*

In the evaluation of a patient's potentiality for postoperative hemorrhage, it is well to obtain a complete history. The faculty of drawing conclusions and inferences as to the operative fitness of the patient increases with experience. The clinical findings and proper laboratory analyses will help the oral surgeon determine the potential operative hemorrhagic risk. Because the entire picture of hemostasis is not understood as yet, it is not possible to arrive at an infallible conclusion, on the basis or normal laboratory and clinical data and history, that the patient will not have an episode of postoperative hemorrhage. There are, however, certain well defined facts and hypotheses which will be helpful in selecting patients for operation. In the light of present knowledge certain conditions can be diagnosed, and the dental surgeon should be aware of these, so that he can anticipate impending difficulty and seek the aid of a physician in evaluating the risk. These conditions may be considered as of a general systemic nature, in contrast to others that are essentially local.

■ *Theory of Coagulation*

The classic concept of coagulation of blood resolves itself into these two equations:

1. Prothrombin + calcium + thromboplastin = thrombin.

2. Fibrinogen + thrombin = fibrin.

Calcium is essential to the conversion of prothrombin to active thrombin. Hemorrhagic conditions due to lack of calcium are not often evident because they would be manifest only when the calcium level was 2.5 mg. per hundred cubic centimeters, with which the evident symptoms of hypoparathyroidism and tetany would be present. The foregoing equations leave many problems unsolved, but they have withstood continued investigation and represent the best analysis achieved to date.

The body is not likely to relegate all the responsibility for hemostasis to the one critically balanced process indicated by the formulas. The organism is economical and efficient, and the vital functions often are protected by multiple mechanisms which can take over each other's functions at least partially. Further, it must be understood that the fine chemical mechanism of coagulation can be utilized for some other purpose. This is suggested by the fact that the blood contains about five times the amount of fibrinogen needed for the effective control of hemorrhage. Fibrin is frequently found in pathologic processes, and its presence must be purposeful. The body can convert fibrinogen to fibrin only with the aid of thrombin, and the coagulation mechanism must be at work in this process.

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Quick[1] summarized the problem in the following statement:

> While the importance of the coagulation mechanism has presumably been underestimated because of the prevalence of the view that it functions only in the defense against hemorrhage, its role in hemostasis has assuredly been greatly overrated. The misconception that the coagulation of the blood is synonymous with hemostasis is responsible for much of the existing confusion. The organism which assiduously protects its vital mechanism is not apt to entrust the task of protection against hemorrhage to one isolated reaction, and it is entirely probable that the coagulation of the blood is only one coordinated part of the coagulation mechanism.

In recent years the importance of the clotting mechanism has received little consideration, and the function and efficiency of the endothelial system have been shown to be of great importance. Many conditions, both local and general, can alter the endothelium, and these must be kept in mind in consideration of hemorrhagic conditions.

Whether there are anticoagulants in the blood stream is controversial. Some authorities[2] have stated that the whole mechanism of coagulation is a delicately balanced antagonistic process, with the anticoagulating factors pitted against the coagulating factors. Others[1] have expressed the belief that anticoagulating factors have not been identified unequivocally. Whether there are anticoagulating factors is probably of only academic interest at this time.

■ *Coagulation Units*

THROMBIN—The activity of thrombin on fibrinogen must be considered the basis of coagulation. Fibrin is the final product of interest in the blood-clotting mechanism, and since fibrinogen is a wholly passive material, thrombin must be the potent force. Thrombin is believed to be an enzyme; apparently little is lost in the process of conversion of fibrinogen to fibrin, and it can be used over and over. Thrombin concentrations in the plasma are controlling factors in rate of coagulation. Sluggishness of the circulation tends to increase the liability to thrombosis.

PROTHROMBIN—Prothrombin, a precursor of thrombin, is found in the blood plasma, and the liver seems to play an important part in its production. Vitamin K is necessary for its production, and there must be some liver function for the production of prothrombin.

THROMBOPLASTIN—A complex material present in platelets and in various tissue cells, thromboplastin is essential to the conversion of prothrombin to thrombin. It is contained in most cells of the body, but its presence seems to be most important in the thrombocytes. It is an intracellular substance, and does not occur free in the blood plasma, as do fibrinogen and prothrombin. This makes it the "trigger" substance and a limiting factor in coagulation. Its liberation actually sets off the physiologic mechanism for coagulation. One of the reasons why the circulating blood does not coagulate is that only by rupture of the platelets is thromboplastin liberated. The foregoing statements are made with some reserva-

1. Quick, A. J., *The Hemorrhagic Diseases and the Physiology of Hemostasis*. Springfield, Ill.: Charles C Thomas, 1942.

2. Tocantins, L. M., The Mechanism of Hemostasis. *Ann. Surg.* 125:292 (March) 1947.

tion because of the newer concepts of hemophilia.

FIBRINOGEN—Fibrinogen is a blood protein found in the plasma which has the ability to gel with a crystalline structure. This makes it an ideal hemostatic agent. The liver is an important factor in its synthesis, although it is generally believed that other factors also are needed. The function of fibrinogen in hemostasis is related to the action on it of thrombin, which determines the quality of the clot formed. A fibrin clot will not stop bleeding unless it is adhesive and produces an adequate seal. Blood flow and pressure tend to dislodge the clot unless it is of good quality and properly anchored.

PLATELETS—Platelets are cellular carrying mechanisms that come from the megakaryocytes of the bone marrow. There is evidence that some come from the liver, the spleen, and the lungs. The platelets are involved in all the mechanisms of the body that control hemostasis. Coagulation is induced by the platelets. They furnish thromboplastin, assist in attaching the fibrin stands to the vessel walls and are responsible for initiating clot retraction.

The most elementary reaction in hemostasis probably occurs with the injury of a capillary. A simple massing of the platelets is the only reaction to this injury, and with the help of low blood pressure, low volume of blood and easily collapsible vessels, this produces a good plug. Under other conditions, massing of platelets is accompanied with swelling of the cells, which ultimately burst and liberate their clot-accelerating agent. The whole mass becomes granular, fixed and adherent to the affected surface. A thin fibrin film then develops between the platelet mass and the flowing blood to form a clot. This process requires an adequate supply of platelets, the proper degree of agglutinability and the capacity for adhesion to certain foreign surfaces. Tocantins[3] reported that clot retraction depends on the platelets and occurs if the count is greater than 70,000 per cubic centimeter.

■ *The Hemostatic Plug*

The effectiveness of the hemostatic plug depends on how promptly it is formed and its ability to meet certain physical requirements. It must have adhesiveness, rigidity and contractibility. All these factors may not appear early in its development, but the speed and quality with which they develop will determine the effectiveness of the plug. It is believed that all the coagulation units are factors in the attainment of these physical requirements. The mode of their activity is not entirely clear.

■ *Vessel Dynamics*

Vessel contractibility and retractibility are among the most important factors in hemostasis. In health, the walls render the vessels distensible and contractile on the basis of their muscle coat. Mechanical injury will produce a response at once either from the vessel itself or over a complex vasomotor pathway. This can be illustrated by the vessel contraction that takes place with the application of cold.

The elasticity of large vessels pro-

3. Tocantins, L. M., Experimental Thrombopenic Purpura in the Dog. *Arch. Path.* 21:69 (Jan.) 1936.

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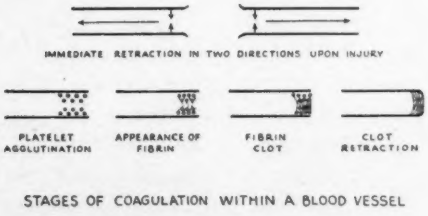
tects them from excessive internal and external stress and aids them in retraction and in reducing their walls when they are injured. The intima rolls to reduce the size of the lumen, while the cut ends of the vessel pull away from each other in contraction. Vessel spasm, contraction and retraction of the vessel wall and the puckering of the intimal coat all lead to the formation of a suitable surface for the accumulation of platelets. Eventually this contraction of vessels ceases, and an adequate plug must be formed by this time or hemorrhage will be resumed.

The location of vessels often puts them at a disadvantage as far as their natural protective mechanism is concerned. Vessels that run parallel to body surfaces are not easily influenced by supporting tissues. Vessels in the mouth not only run parallel to mucous membrane surface but on one surface have only a ridged bony support, which is incapable of response. This anatomic situation may be influential in the delay of hemostasis. Regions in which vessels lie close to the surface—for example, the mouth—are especially likely to hemorrhage when a hemorrhagic disorder is present.

The degree of tension of tissue over vessels has a controlling influence on the ability of the vessels to aid hemostasis. Bleeding from small abrasions or cuts on the skin or mucous membrane ceases before there has been any considerable loss of blood when the tissue closes over the wound and the elementary process of capillary response comes into play. The integument acts as a protective cover over the open vessel. It is possible that the saliva may act as a minimal seal to abrasions of the oral cavity. The converse of the help the supporting tissue gives in hemostasis is exemplified by the difficulty in control of hemorrhage from the cut surface of bone.



STAGES OF COAGULATION WITHIN A BLOOD VESSEL

Fig. 1.

■ *The Method of Hemostasis*

When a vessel is cut, its internal blood pressure is reduced immediately, and its walls collapse. The additional pressure in the surrounding tissues increases tissue tension and prevents further flow. The increase in tissue pressure tends to force blood through other channels and reduces the pressure in the region of the injury. Contraction of vessel walls with occlusion of the lumen may last for days, but usually lasts only a few minutes.

The slowing of the blood by vascular activity allows the blood to come in contact with the extravascular tissues and the damaged vessel walls. This leads to the second phase of the process, the massing of the platelets. The longer the first phase lasts, the greater the chance for the second phase, and the greater the possibility of success in accomplishment of hemostasis. The process in large vessels is shown graphically in Figure 1.

Determination of the site of the defect in the hemostatic mechanism serves as a basis for the clinical approach to the

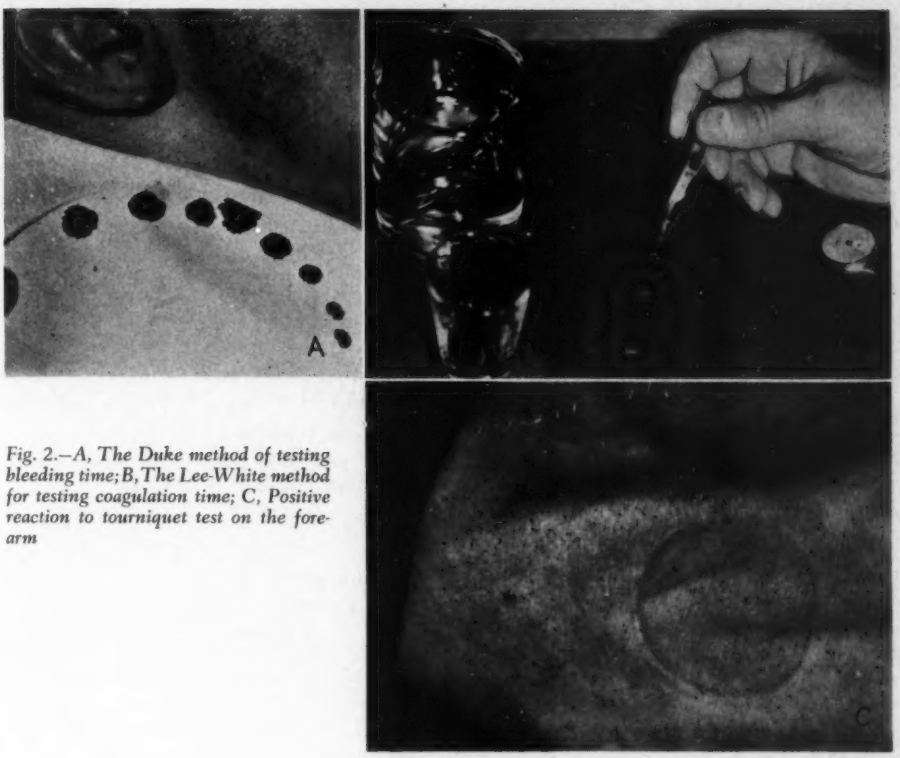


Fig. 2.—A, The Duke method of testing bleeding time; B, The Lee-White method for testing coagulation time; C, Positive reaction to tourniquet test on the forearm

problem of differential diagnosis and treatment of all hemorrhagic syndromes. Laboratory procedures for evaluation of the blood are numerous and complex. A few that are of tremendous value may be completed with a minimum of effort and equipment. Of these, the red cell count, white cell count, hemoglobin determination, differential count and platelet estimation comprise the general routine. Additional information can be gained by four other relatively easy tests: determinations of bleeding, clotting and clot retraction time and the capillary fragility test.

BLEEDING TIME—The Duke test[4] is performed with a minimum of equipment (Fig. 2,*A*). An incision about 2 mm. long is made in the lobe of the ear with a small sharp blade. The tissues are separated, if necessary, to allow free bleeding. The drops that form from the lobe are collected without pressure every thirty seconds on a filter paper. The period of bleeding is timed. Normally, the drops become progressively smaller and cease

4. Duke, W. W., The Relation of Blood Platelets to Hemorrhagic Disease: Description of a Method for Determining the Bleeding Time and Coagulation Time and Report of Three Cases of Hemorrhagic Disease Relieved by Transfusion. *J.A.M.A.* 55:1185 (Oct. 1) 1910.

forming in approximately five minutes. A normal range is from three to eight minutes. If bleeding continues longer than twenty minutes, cotton should be applied with a little pressure to stop the hemorrhage.

Bleeding time gives the best index of capillary contractibility. It is influenced by the permeability of the vascular walls and perhaps by the platelet count, but these factors can be determined separately and their importance weighed. The ruddy person often has a somewhat prolonged bleeding time. If other data are within normal limits and the patient has had no special difficulty with bleeding, determinations of bleeding time should be made at intervals of several days. If there is no change, the prolongation can be considered insignificant.

COAGULATION, OR CLOTTING, TIME—Because blood clotting is influenced by temperature, size of drop and the smoothness and cleanliness of instruments, the coagulation time can best be measured with blood taken from the vein. The Lee-White method[5] is probably the most reliable. In addition, this method offers a test for clot retraction time which can be done easily at the same time. A 10 cc. syringe and 3 scrupulously clean serologic tubes about 8 mm. in diameter are rinsed with physiologic solution of sodium chloride. About 5 cc. of blood is withdrawn from a vein with a minimum of stasis, and 1.5 to 2.0 cc. is placed in each tube. The time when the blood first appears in the syringe is recorded. After three minutes, one of the tubes is tilted at one minute intervals until it can be inverted without the blood being dislodged. This is repeated with the second tube until it also can be inverted. The total time consumed is the coagulation time. The normal value is from six to twelve minutes.

The third tube can be used to determine clot retraction time. A stopper is inserted and the tube placed in a water bath or incubator at body temperature. It is inspected at one, two and three hour intervals, and, if retraction is still deficient, after twenty-four hours. Normally there should be good retraction at the end of an hour. Figure 2,*B* shows a setup for this procedure.

If impairment of coagulation time is found, it is logical to infer that there is a defect in the prothrombin-calcium-fibrinogen-thromboplastin mechanism. Tests for calcium and fibrinogen are seldom necessary, because deficiencies in these materials are rarely found in clinical situations. If the history and clinical picture do not suggest hemophilia, it would be well to enlist laboratory aid and perform a prothrombin determination. The general appearance of the patient and an evalutaion of the results of the various tests are helpful in determining the need for the prothrombin test.

The phenomenon of clot retraction apparently is parallel to the number of platelets. It is entirely independent of the coagulation time. This can be shown best in the presence of purpura haemorrhagica, in which the coagulum is formed within normal limits, but the clot retracts little or not at all after standing several days. In hemophilia, with a normal number of platelets, the blood coagu-

5. Todd, J. C., and Sanford, A. H., *Clinical Diagnosis by Laboratory Methods: A Working Manual of Clinical Pathology*. Philade'phia: W. B. Saunders Company, 1944, pp. 197-199.

lates slowly, but the clot retracts normally. These facts are applicable to women with prolonged menses who are afflicted with purpura. In these a bleeding tendency is suspected, and the only confirming factors are the positive tourniquet test and delayed retraction. Coagulation time in this instance is inconclusive.

CAPILLARY RESISTANCE (TOURNIQUET TEST)—By the tourniquet test it is possible to evaluate the ability of the capillaries to resist injury. A blood pressure cuff is applied above the elbow and the systolic and the diastolic pressure determined. A point halfway between these two pressures, but in no instance greater than 90, is chosen as the pressure to be applied. This pressure should be held for five minutes. If petechiae are produced, a hemorrhagic disorder is indicated. Figure 2,C shows the method and a positive test result.

It must be kept in mind that a positive result can be obtained for a normal subject if the procedure is not judiciously carried out. Normal vessels have a point at which they will be overcome by pressure and will fenestrate.

To determine the degree of bleeding to be anticipated from operation, the history, clinical symptoms and results of the various clinical tests must be evaluated. Abnormalities suggest consultation with the patient's physician.

■ *The Hemorrhagic Conditions*

History and laboratory studies give strong indications of many of the abnormalities of hemostasis, and one can profit from a review of these entities (see Table).

BLEEDING TIME PROLONGED; COAGULATION TIME NORMAL—A. Hereditary Pseudohemophilia. Pseudohemophilia is a hereditary hemorrhagic disease transmitted by and appearing in patients of both sexes. There is no contraction of the capillaries when they are punctured. The disease is characterized by prolongation of bleeding time, and no other abnormality of the blood or the coagulation mechanism is demonstrable. A positive reaction to the tourniquet test is common, and the tendency to bruise and bleed easily is the only sign of the mild form. However, in the severe form there is a tendency to frequent and severe epistaxis and the appearance of large ecchymoses spontaneously or from mild trauma. The disease manifests itself in infancy. It has been stated that pseudohemophilia is as common as true hemophilia. Patients with this disorder are extremely poor surgical risks, because the defect seems to be in the retraction of the vessels, and transfusion is of relatively little value. Troublesome bleeding is common after the extraction of teeth and may occur even in children with the loss of deciduous teeth. Local treatment is the best that can be offered at this time.

B. Purpura. Purpura is a symptom complex whose name comes from the Greek word meaning purple. The condition is characterized by hemorrhages beneath the skin and mucous membranes, frequently associated with bleeding from the orifices of the body. Usually the bleeding is rather close to the body surfaces. Purpuric spots often are seen around the mouth and associated structures (Fig. 3), and often hemorrhage persists after minor oral surgical procedures. The condition

is common to all ages and twice as common in female as in male patients. The important changes that characterize the condition are: (1) deficiencies in blood coagulation, (2) an increase in fragility and permeability of the capillaries and (3) a combination of these.

Coagulation may be impaired by arrested maturity of the megakaryocytes, a reduction in the formation of the platelets or the destruction of the platelets in the blood stream. According to Tidy,[6] the platelets serve as a seal for capillaries and, by deposition on the capillary walls, seal the endothelium against the escape of the blood into the tissues. In some instances a large number of platelets may be needed to seal the vessel walls, and even when cells are normal in both number and structure, they may not be sufficient if the vessels are highly permeable. This may be the case in hereditary telangiectasia, anaphylactoid purpura and purpura fulminans, and will explain why prolonged bleeding time may occur when coagulation time is adequate, which would indicate that platelets are sufficient for the task at hand.

Purpura is classified as primary idiopathic (the cause of which is unknown), and secondary (in which an etiologic factor can be found). There is another form in which part of the diagnostic picture is present but platelet activity is normal. The diagnosis is based largely on prolonged bleeding time and a positive tour-

6. Tidy, H. L., Haemorrhagic Diathesis: Angiostaxis. *Lancet* 2:365 (Aug. 21) 1926.

The hemorrhagic conditions

Condition	Sex Limitation	Platelets	Bleeding time	Coagulation time	Clot retraction time	Prothrombin time	Capillary resistance	Hereditary factor
Hereditary pseudohemophilia		Normal	Prolonged	Normal	Normal	Normal	Positive	Yes
Purpura Thrombocytopenic		Low	Prolonged	Normal	Poor	Normal	Positive	No
Nonthrombocytopenic		Normal (?)	Prolonged	Normal	Normal	Normal	Positive	No
Hypoprothrombinemia		Normal	Normal	Normal or prolonged	Normal	Reduced	Negative	No
Hemophilia	Male	Normal	Normal	Prolonged	Normal	Normal	Negative	Yes
Hereditary hemorrhagic telangiectasia	Female 2–1	Normal	Normal (?)	Normal	Normal	Normal	Negative	Yes
Polycythemia		High	Normal or prolonged	Normal	Poor (?)	Normal	Negative	No
Vitamin C deficiency		Normal	Normal	Normal	Normal	Normal	Positive (?)	No

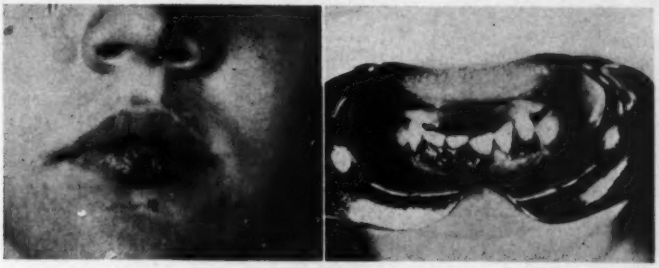


Fig. 3.—Left: purpuric spots on the lips. Right: large purpuric areas on the gingiva

niquet test. Syneresis is delayed or absent and is not constant. In the conditions with known cause, treatment has been successful.

COAGULATION TIME DELAYED; BLEEDING TIME NORMAL.—A. Diminished Prothrombin. One of the causes of diminished prothrombin has been found to be lack of vitamin K, which is essential for the formation of prothrombin in the liver. The coagulation time is not greatly affected until liver function has been reduced to 20 per cent. Further, it must be kept in mind that some liver function must be present for formation of prothrombin.

A condition known as hemorrhagic disease of the newborn has been recognized for centuries. In the normal child the prothrombin level is about normal at birth, falls on the third or fourth day and rises again to about normal on the seventh or eighth day. Hemorrhage at this time may be inconsequential or of great magnitude, causing damage to the internal organs and the brain and even death from exsanguination. Prevention of this condition is accomplished by the administration of vitamin K to the mother for a period before birth or to the child at birth and until the vitamin K level is assure. The cause of this condition is believed to be a lack of the gastrointestinal flora necessary for the formation and absorption of the vitamin K for the first few days.

In the adult a similar picture is found (1) in obstructive jaundice because of impaired absorption of vitamin K, (2) in advanced liver disease because of failure in formation of prothrombin, (3) in disturbance of the absorptive surfaces of the intestine in which the prothrombin is not picked up and (4) in possible dietary deficiency in vitamin K. This vitamin is one of the fat-soluble group, and bile salts are essential for its absorption in the intestine. Keep this in mind when the material is administered, and the salt given in indicated doses.

It is known that hypoprothrombinemia may result from large doses of salicylates. Recently some investigation has been reported by Cahn[7] and Rapp,[8] indi-

7. Cahn, Lester, Acetylsalicylic Acid (Aspirin) as a Cause of Bleeding. *J. Oral Surg.* 3:336 (Oct.) 1945.

8. Rapp, G. W., A Cause of Delayed Hemorrhage after Tooth Extraction. *J.A.D.A.* 34:484 (April 1) 1947.

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cating that rather small oral doses may produce this condition. It is suggested that when salicylates are given postoperatively, vitamin K be added. With this preparation any drop in prothrombin level can be prevented. Another drug to be considered in the prevention and control of postoperative thrombosis is dicumarol.[2] This material has the ability to cause a drop in prothrombin level and may produce capillary dilatation resulting in undesirable hemorrhage.

The patient with jaundice often has no history of episodes of hemorrhage, but usually any such history is associated with the postoperative course of a previous trauma. Before the patient with jaundice can undergo surgical treatment, the prothrombin level must be raised.

B. Decreased Fibrinogen. A decrease in fibrinogen content is rare. The condition sometimes is hereditary and sometimes occurs with severe liver disease. It is the only hemorrhagic condition, except acute yellow atrophy, in which the blood remains absolutely and permanently incoagulable. It is of little clinical importance, because fewer than a dozen cases have been reported.

C. Hereditary Hemophilia. Hemophilia is one of the most dramatic diseases known to man. It is a sex-linked disease, manifested in the male and transmitted through the female. It usually makes its appearance in the first or second year of life, and few hemophiliacs survive infancy. The patient with hemophilia must be protected against all forms of injury, and any bleeding becomes a serious thing. The presence of hemophilia usually can be suspected without clinical studies because of the past history, and generally the patient is aware of his condition by the time he appears for dental treatment. The history usually will reveal evidences of hematuria and of hemarthrosis. Often the latter condition can be suspected from the gait. The delay in clotting in this condition has been attributed to the inability of the platelets to break down and release their thromboplastin.[9] Recent work has indicated that this explanation may not cover the situation. Patek and Stetson[10] showed that if plasma is free from formed elements, including platelets, coagulation time is reduced. A series of reports from the Thorndike Memorial Laboratories[11] over the past twelve years has shown that the antihemophilic property of platelet-free plasma is closely associated with the globulin fraction of the protein and that the antihemophilic factor is neither prothrombin nor fibrinogen. Globulin fractions free from both prothrombin and fibrinogen were prepared from normal human plasma. These had optimal effect in reducing the coagulation time of the hemophiliac to normal. An antihemophilic globulin has been produced and is available for use. It has shown promise of being helpful in some instances, although it has not been entirely successful in control of hemophilic hemorrhage.

The clinical picture of hemophilia should be clear from these factors: (1) bleeding (deep as contrasted with that in

9. Minot, G. R., and Lee, R. I., The Blood Platelets in Hemophilia. *Arch. Int. Med.* 17:474, 1916.

10. Patek, A. J., Jr., and Stetson, R. P., Hemophilia: Abnormal Coagulation of Blood and Its Relation to Blood Platelets. *J. Clin. Investigation* 15:531 (Sept.) 1936.

11. Minot, G. R., and Taylor, F. H. L., Hemophilia: The Clinical Use of Antihemophilic Globulin. *Ann. Int. Med.* 26:363 (March) 1947.

thrombopenic purpura), (2) sex incidence (affecting only the male) and (3) hereditary aspects (transmitted through female to male). The platelet count is normal and the bleeding time normal or slightly prolonged. The coagulation time may range from eighteen minutes to many hours. Prothrombin and clot retraction time and capillary resistance are within normal limits.

Success has been reported in treatment of hemophiliacs who need extraction of teeth. Unless some of the new materials offer help, the outcome of treatment must be weighed against the possible loss of life. The ability to clot seems to vary greatly from time to time, and with no better knowledge than is now available, the hazards are great. Preoperative and postoperative transfusions of whole blood and plasma are beneficial. When hemorrhage is present, local application of pressure may help. The direct application of fibrin foam or absorbable cellulose with heavy concentrations of thrombin[12] will help.

Whenever it becomes necessary to operate on a hemophiliac, it must be kept in mind that a cleancut puncture wound will be easier to control than a lacerated wound. If a puncture wound must be made for blood study, it should be made on a finger, where adequate pressure can be applied to stop resultant hemorrhage. Extractions sometimes can be done by the ligature method, but often the teeth are badly broken down and do not allow traction. If other methods are used, great caution should be used after evaluation of the clotting time and suitable preparation with plasma, whole blood or antihemophilic globulin.

BLEEDING AND COAGULATION TIMES NORMAL—Hereditary Hemorrhagic Telangiectasia. Hemorrhagic telangiectasia is an inherent familial maldevelopment of the minute blood vessels in localized areas that predisposes them to serious bleeding on slight injury. The condition occurs in both sexes and manifests itself in middle life, most commonly as epistaxis. Telangiectasia often is seen around the mouth and other parts of the face. The disease carries the triad of heredity, hemorrhage and angioma. The bleeding and clotting times are normal, and the most suspicious finding is the local character of the hemorrhage. This condition is not difficult to treat from a surgical standpoint, because the hemorrhages are local and can be treated locally when encountered.

■ *Other Conditions Associated With Hemorrhagic Tendencies*

The capillary walls are altered in certain miscellaneous conditions. Vitamin C deficiency should be considered. Although the condition is rare in adults in the United States, cases are reported from time to time, and the existence of a subclinical state must be considered. The importance of vitamin C to the endothelial system is well known. In association with this vitamin is vitamin P, which is believed to be important in maintaining capillary resistance and permeability. In vitamin B deficiency, bleeding with slight traum also is characteristic. Often infectious gingivitis is superimposed on an already existent disease. In diabetic stoma-

12. Losch, P. K., The Use of Fibrin Foam with Thrombin in Dental Operations. *J. Oral Surg.* 3:177 (July) 1945.

titis the tissues have poor tone and bleed easily. Among conditions that show reduced capillary resistance, Madison and Squiers[13] listed the following: (1) vitamin C deficiencies; (2) severe infections (scarlet fever, pneumonia, bacteremia, subacute bacterial endocarditis); (3) malignant disease (terminal stage); (4) malignant hypertension; (5) primary blood disease (leukemia, multiple myeloma, aplastic anemia, purpura); (6) allergic reactions; (7) ovarian dysfunction.

Polycythemia is a chronic generalized condition of insidious onset. The facies and mucous membranes are dark red. The red blood cell count is above 7,000,000, and thrombocytosis is evident. There is a tendency to bleed postoperatively. This can be explained best on the basis of the high viscosity of the blood and its high content of solid material, which place a strain on the vessels and on the clot formed. Hemorrhage in this disease can best be controlled on a local basis and is not too serious. Phlebotomy is done for treatment of the condition.

Conditions which tend to allow the destruction or retard the formation of the blood elements will manifest themselves with hemorrhage in the oral cavity. Some such conditions are pernicious anemia, aplastic anemia and leukemia (a moderate increase in bleeding time plus thrombopenia may be found). Henny[14] stated: "It must be kept in mind that most of the types of dyscrasia that produce oral lesions also predispose the patient to dangerous hemorrhage."

In hemorrhage resulting from conditions of a more or less general character, it is important to keep in mind the diagnostic points, so that operation will not be undertaken until adequate preparation has been made. The patient with hemorrhage from a local cause can be treated on an entirely different basis from the patient with a general hemorrhagic condition. No patient suffering from a blood dyscrasia that predisposes to hemorrhage should be operated on until the true nature of the condition has been determined and adequate treatment and hospital care provided.

■ *Control of Local Hemorrhage*

The great majority of hemorrhagic problems are from local causes. Local hemorrhages have been classified clinically on a basis of time: (1) primary or immediate; (2) intermediate, that is, occurring within a few hours or resulting from a dislodged clot, and (3) secondary, occurring after forty-eight hours or more and due usually to infection or slough. In any case of severe hemorrhage, it is imperative to control the bleeding from the large vessels at the outset. Application of pressure to points known to carry the large vessels is helpful. Around the head and neck the superficial temporal, external maxillary and carotid arteries can be used. Arterial bleeding can be recognized by pulsation and can be controlled best by pressure on the vessels close to the heart. Venous blood is slightly less red than arterial blood and oozes forth gradually. The use of pressure packs is more effective for venous than for arterial hemorrhage. Tracing the course of veins is difficult and when no valves are present is of little value.

13. Madison, F. W., and Squier, T. L., Etiologic Factors in Hemorrhagic States. *Wisconsin M. J.* 37:636 (Aug.) 1938.

14. Henny, F. A., Significance of Blood Dyscrasia in Oral Surgery. *J. Oral Surg.* 2:106 (April) 1944.

After the initial control, a more definitive hemostasis should be accomplished. It must be kept in mind that hemorrhage should be stopped with a minimum of trauma and of foreign body inclusion. All bleeding from moderate and large vessels must be adequately controlled. The decision as to whether small vessels should be tied must rest on the operator's belief as to whether final closure will establish adequate hemostasis from secondary hemorrhage. When there is severe bleeding from bone, a blunt instrument should be used to burnish the bone at the point of bleeding. Occasionally, the burnishing of bone must be augmented by application of "bone wax" to the site and burnishing in the manner used for burnishing trabeculas. A satisfactory formula for bone wax is Horsley's, which consists of 1 part phenol crystals, 2 parts olive oil and 7 parts yellow wax.

Arterial bleeding must be controlled before any other. If the bleeding point can be defined, a hemostat of appropriate size can be closed on the vessel, care being taken that only the vessel is caught, since the more tissue included, the more slough will result. Vessels should be ligated at both the distal and the proximal end, so that when collateral circulation is established there will be no additional bleeding from this region. Troublesome venous bleeding often can be handled in the same way. Ligatures of silk, cotton or catgut can be used. They should be large enough so that increases in pressure will not cause rupture but not so large as to create unnecessary foreign body reaction. Ligatures should not be tied so tightly that they will cut the intima. All vessels should be tied firmly but not cut by the suture.

Cautery is occasionally valuable in the control of bleeding, especially when the affected site is deep and sutures are hard to apply. It is effective in limiting infected processes and controlling hemorrhage when infection is present. A red hot instrument may be used to touch sites of persistent bleeding. It is well to keep the heat at a minimum so that the tissue is not cut through. The method is speedy and effective for small vessels, but may leave carbonized tissue in the wound to act as a foreign body.

Occasionally in the course of an operation a portion of tissue can be so placed as to act as a compressor of a vessel and produce hemostasis. General placement of the body aids in control of bleeding around the head and neck. Any method of decreasing the blood pressure to the part concerned is valuable.

Consideration must be given to temperature and its local effect on this problem. Applications of cold to the part must be considered to be effective, since they have a tendency to constrict blood vessels. However, when the wound is open, hot packs may be applied to the immediate site for their increase in the speed of coagulation of the blood.

A common point of hemorrhage often is the site of tooth extraction. Careful surgical technic will reduce the amount of postoperative hemorrhage. Cleancut wound margins will present less difficulty than ragged edges. Forcing the alveolus back into its former position after it has been sprung away in the operation will help to close bone vessels. Readaptation of margins over the wound and tack-

ing them to position with a few sutures will aid in control of bleeding in soft tissue. Immediately after the operation is complete, it is well to establish some definite and continued pressure over the wound. This can be accomplished by placing a sponge or other piece of gauze over the site and having the patient apply biting pressure for twenty minutes. When the patient leaves the office, he is told not to rinse the mouth until the next day. The patient who feels he must spit out any slightly blood-tinged fluid should be especially warned. He will return home and sit by the toilet or with a receptacle and expectorate all the fluid that comes into his mouth. Every time he relieves himself of some of this material, he will create a suction by the activity of the buccinator muscle over the wound, and thus encourage continued bleeding. He should be instructed to take a roll of gauze and apply pressure if bleeding persists or is excessive. If this is ineffective he should call the dentist for further instructions.

The hemorrhage following tooth extraction usually ceases in good order and within a few hours spontaneously. Occasionally it does not, and in some cases of intermediate or secondary hemorrhage there may be a foreign body at the extraction site. If roentgen examination shows no evidence of a foreign body, the wound should be examined. The use of procaine hydrochloride as an anesthetic usually is a definite aid to the condition because of its vascoconstricting agent. However, if the dentist considers a general anesthetic advisable, the addition of a little local anesthetic to the site after the patient has been anesthetized will be valuable in immediate slowing of the hemorrhage.

The socket should be carefully cleaned of all residual clot and the alveolus and soft tissues scrutinized for the site of the bleeding. If there are definite bleeding vessels, they can be tied off; if they are in bone, burnishing of some of the trabeculas should be sufficient treatment. If no bleeding vessels are evident, which usually is the case, many means of hemostasis are available. One of the most favored materials is solution of ferric subsulfate. It has been reported that the material is not especially suitable, since it allows the clot to come away from the vessel easily so that bleeding will start again. Surgeons who have used it attest also that it makes the field black and may discolor most of the mouth. Epinephrine applied to the area on a sponge or pack is effective, although its activity is of short duration. Often when hemorrhaging spots are rather soft and spongy and contain much granulation tissue, the monopolar electrothermic current may be set for coagulation and passed to the sites under treatment.

A new hemostatic agent has come into common use recently. This material is for local application and is specific for local clot formation. As was previously stated, there is an abundance of fibrinogen in the blood. It is evident, then, that if the defect in clot formation is related directly to the clotting mechanism, it must be based on lack of thrombin. Thrombin has been proved to be the most powerful of all clotting agents. It combines with soluble fibrinogen almost instantaneously to precipitate the crystalline fibrin of the blood clot. Thrombin has been known for years to be effective, but its surgical

use has been possible only recently, because means of extraction of the active principle in adequate potency and of assuring the bacterial sterility of the product now have been discovered. This material is an important addition to the group of hemostatic agents, but cannot be expected to handle all the problems because their causes are varied.

Absorbable sponge materials now are available for use with thrombin. These substances were developed as a result of development of blood fractionation products during the war. A material produced from a blood fraction and called fibrin foam[15] has been used for some time and has proved wholly successful in treatment of hemorrhage. The reaction of the tissues to buried fibrin foam is little more marked than the reaction to absorption of a simple hematoma of comparable size.

Fibrin foam, being produced from human blood fractionation, has limits on its availability, and its cost is high. One substitute has been manufactured from animal gelatin, which is plentiful and cheap. This material, a gelatin sponge,[16] is the equal of fibrin foam in hemostatic properties, is less likely to crumble and can be cut easily.[17] When wet it maintains high tensile strength and is easy to press into a wound site. It should be borne in mind that gelatin *per se* is an extremely good culture medium for bacteria, and the introduction into contaminated wounds of a substance which will aid the growth of bacteria has its limitations.

Oxidized cellulose[18] is another hemostatic agent which has application in control of surgical bleeding. This material is highly acid, and unless it is soaked in a 1 per cent solution of sodium bicarbonate or used immediately, it will nullify the activity of the neutral thrombin. Oxidized cellulose has been found to be effective when used without thrombin. In itself, it has a certain styptic property which may have some merit. Frantz and Lattes[18] stated:

> The only untoward effect, apparently, when the material is introduced into the tissues, is in bone. The acidity of the gauze is considered the probable cause of delay in the formation of the early callus in clean experimental fractures and defects. Ultimately, however, repair does take place, and there is, of course, no significant delay when, in the open lacerated compound fracture, hemostasis is secured by this means and the gauze is removed in the course of one to three days. Some retardation of epithelization has also been observed, and oxidized gauze is not recommended for a surface dressing of open wounds, except for hemostasis, to be removed when it has served this purpose. In this connection it is well to place it so that it does not overlap the skin edges, where it will stick like dry glue in contrast to its jelly-like character against moist tissue. It is this property of becoming gelatinous in the tissue fluid which allows it to be removed from the open wound without starting fresh bleeding and which also is insurance against any possibility of blocking drainage.

Without thrombin this material is of value only in stemming minor bleeding, and severe hemorrhages should be treated with the addition of thrombin.

15. Ingraham, F. D., and Bailey, O. T., The Use of Products Prepared from Human Fibrinogen and Human Thrombin in Neurosurgery: Fibrin Foams as Hemostatic Agents: Fibrin Films in Repair of Dural Defects and in Prevention of Meningocerebral Adhesions. *J. Neurosurg.* 1:23 (Jan.) 1944.

16. Light, R. U., and Prentice, H. R., Gelatin Sponge: Surgical Investigation of New Matrix Used in Conjunction with Thrombin in Hemostasis. *Arch. Surg.* 51:69 (Sept.) 1945.

17. Pilcher, C., and Meacham, W. F., Absorbable Gelatin Sponge and Thrombin for Hemostasis in Neurosurgery: Experimental and Clinical Observations. *Surg., Gynec. & Obst.* 81:365 (Oct.) 1945.

18. Frantz, V. K., and Lattes, Raffae'e, Oxidized Cellulose—Absorbable Gauze. *J.A.M.A.* 129:798 (Nov. 17) 1945.

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These materials should be applied judiciously to a bleeding surface. The material should be cut under sterile conditions into rather small pieces and placed in extraction sites without any attempt to fill the defect. The material tends to swell and, if too much is used, will tend to force the tissues from approximation.

All these materials are well tolerated and will be almost completely absorbed in thirty to forty-five days. However, they must be considered to be foreign bodies for many days, and no doubt they act much as absorbable sutures do during that period. Minimum amounts of these materials should be used at all times, and if large quantities are needed for adequate hemostasis, drainage must be established to care for foreign body sequelae.

Many reports in the literature have indicated that these materials are efficacious in treatment of postextraction hemorrhage in hemophiliacs.[12] It is well to keep in mind that they are effective locally but offer nothing but empiricism as far as the general condition is concerned.

Mitchell[19] reported the use of a fibrinogen fraction and thrombin in tooth sockets to form an early artificial clot. Too little work has been reported to justify evaluation of the method, but apparently too little consideration has been given to the endothelial system, which is known to play a large part in hemostasis.

■ *Summary*

1. The diagnosis of various hemorrhagic conditions is based on determination of the defect in the hemostatic mechanism which is responsible for the hemorrhagic manifestations.

2. Defects in hemostatic mechanism can be evaluated with considerable accuracy by the use of simple tests such as formed element counts, determinations of coagulation, bleeding and clot retraction time and the tourniquet test. When indicated, the assistance of the laboratory can be procured for determining prothrombin time.

3. The various methods of hemostasis are evaluated, and their limitations are recognized. Thrombin is a valuable adjunct to other means of hemostasis.

4. With careful attention to history, clinical findings and laboratory data, serious postoperative hemorrhage can be prevented except in the hemophiliac and the pseudohemophiliac, who must be handled with great caution.

5. In the presence of hemorrhage, the specific situation must be analyzed and the applicable hemostatic method selected.—*1424 Grand Rapids National Bank Building.*

19. Mitchell, D. F., Products of Human Blood Fractionation and Their Uses in Dentistry. *J.A.D.A.* 33:1287 (Oct. 1) 1946.

Diagnosis and surgical removal of the mandibular impacted third molar

■ *H. W. Sorensen, D.D.S., San Francisco*

The removal of the impacted mandibular third molar presents a wide variety of problems and complications. It is important that sound judgment be used in the preoperative diagnosis for surgery, in selecting the technic for removal and in the postoperative treatment. This leads to the best end results.

■ *When Should the Third Molar Be Removed?*

The first questions which confront us are these: How early in life and under what circumstances should the unerupted third molar be removed, and when should impacted mandibular third molars in the adult be removed? There has been much discussion as to whether the third molar in the child stimulates the growth of the mandible. It has been proved by Brodie and other investigators that it does not. The unerupted third molar seems to be the cause of the buckling of the incisor teeth in some cases.

The orthodontist has learned that in order to complete and maintain treatment satisfactorily he must keep the teeth on the arch. In every case in which there is too much buccal expansion of the molar and the bicuspids, and labial expansion of the incisors, there will be drifting or buckling after the retainers are removed. Orthodontists now are concentrating on arch length. The problem is to make room for 28 teeth on the arch. If this is not possible, then the permanent bicuspid often is sacrificed or room is made by the distal movement of the mandibular posterior teeth, or by a combination of both. It is easier to move posterior teeth distally with the unerupted third molar out of the way.

Some other indications for the removal of unerupted third molars at an early age are:

1. When all molars are abnormally large.
2. When the second molar is crowded under the convexity of the first molar.
3. When the mandibular third molar is large and the maxillary third molar is small or absent.
4. When the permanent molars tilt forward owing to early loss of the deciduous molars, and these teeth must be tilted back into position.

As for the second question, "What impacted mandibular molars should be removed for the adult?", in almost every case an impacted mandibular third molar should be removed as soon as it is discovered. Preventive dentistry could be practiced in this way. Almost always these teeth, unless removed, are the cause of trouble in later life. They are easier to

Read at the thirtieth annual meeting of the American Society of Oral Surgeons, Chicago, September 9, 1948.

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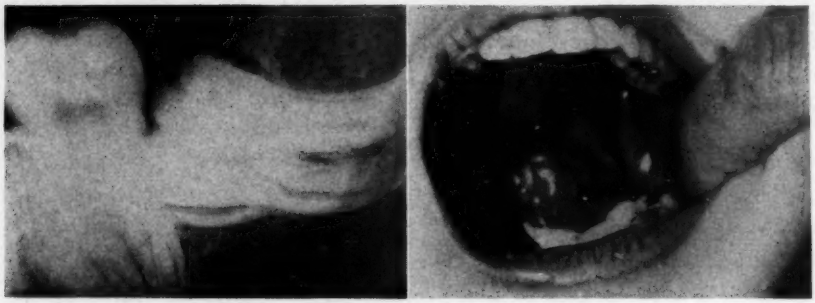

Fig. 1.—Left: Roentgenogram of a horizontal mandibular molar. Right: Rubber mouth prop and gauze sponge in position

remove in early adult life, simplifying the process for surgeon and patient alike, and the period of recovery is shorter. If left undisturbed, in many cases these teeth will cause a destruction of the bone and formation of pockets. They may cause decay and resorption of the adjoining teeth, and pericoronitis with its sequela.

Very little is known about the complicated anastomosis of the fine filaments of the nervous system, but there are at least some grounds for surmise that disease in these teeth may cause many obscure pains in the parietal, occipital and laryngeal regions, often affecting the eyes and many other parts of the body.

■ *Roentgenographic Diagnosis*

In diagnosis one must be able to differentiate between impactions which present extreme difficulties in extraction and those in which the operation will be relatively easy. An adequate roentgenogram and proper reading of it is the greatest help. If the roentgenogram shows the first and second molars without overlapping contact points, and not foreshortened or elongated, then one may expect to see the true relation of the third molar to the long axis of the second molar. Most classifications of the third molar start at this point, as either a perpendicular, horizontal (Fig. 1, left), mesioangular or distoangular impaction. Some are also in lingual or buccal version or present other complications which can lead to a difficult operation.

The roots in every case are different and must be considered factors in diagnosis and operation. The depth of the tooth in the bone may cause the root to impinge on the inferior dental nerve, or the nerve may pass through the roots. The latter condition usually can be diagnosed by study of roentgenograms, as the inferior dental canal will narrow down to about the size of the lead in a pencil. Often the follicular space is filled with cementum or bone, or the bone is extremely dense around the tooth. The most difficult tooth to remove is one in which the crown seemingly has been absorbed by osteoclastic activity and this space has been filled with bone.

If good roentgenograms have been made and properly studied, the removal of the impacted tooth is greatly simplified.

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Each operator bases his technic on training and personal experience. Some use Winters' technic and hand pressure, others burs, others the automatic chisel and others the chisel and mallet. I prefer the chisel and mallet and will give my reasons for preferring this method.

■ *Operative Procedure*

The concepts and principles of good general surgery apply equally to oral surgery. Asepsis is the most important single consideration. Proper planning seems to be next. Speed, as in general surgery, is of next importance; but one should "make haste slowly" and not sacrifice thoroughness or cause undue trauma. The principal concern of this paper is to show a technic for the rapid, easy removal of the average impaction, which embraces about 90 per cent of impacted teeth.

I prefer to operate either in the office, using block anesthesia, or in the hospital, employing a general anesthetic of choice. Always in the hospital and sometimes in the office the patient is given premedication. Whether a patient is given premedication in the office depends on his mental attitude and the confidence he has in the operator. If the patient has four impactions, all four are removed at one time, regardless of whether the operation is done in the hospital or in the office.

In office operations, after block anesthesia the patient's mouth is packed with a gauze roll under the tongue to keep the operating field as dry as possible. A rubber mouth prop is inserted to stabilize the jaw, giving the patient greater comfort and the operator steadiness and room to work. (Fig. 1, right).

The mucoperiosteum is retracted, always cutting to the bone, with the cut made to give good access and at the same time to provide a flap that can be sutured over sound bone. The retractor should hold the flap firmly and be shaped to hold the cheek, to allow good vision without excessive stretching of the corner of the patient's mouth and to avoid undue fa-

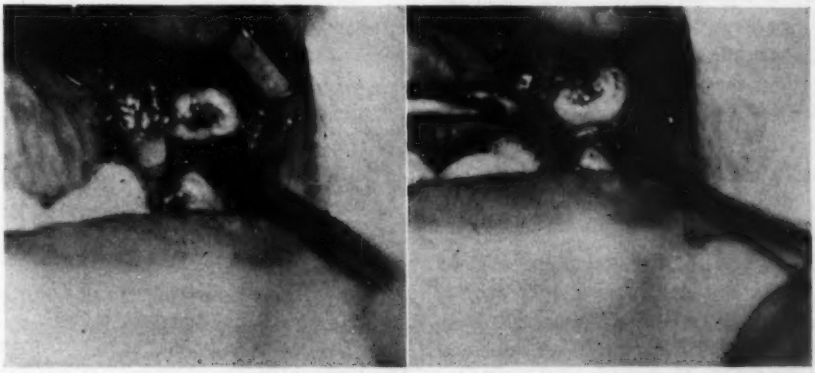

Fig. 2.—Left: Process is removed and third molar is split, with an elevator removing the distal half. Right: Mesial half of molar being elevated

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Fig. 3.—An example of a perfect horizontal split

tigue of the operator in retracting.

The chisels used should be narrow, not more than 3 mm. wide, with a long bevel, and sharp. The harder the temper of the chisel, the harder it is to drive through bone. Extremely hard chisels seem to dull quickly, probably because the bone is hard enough to cause fine chipping of the cutting edge; softer chisels do not dull as quickly in the bone. The temper should be of such hardness that the chisel can be sharpened by a file. Sharp, narrow chisels produce a minimum of trauma. With them there is no possible burning of the bone; the bone fragments are easier to remove than small debris and bone cutting paste; there is no possible element of contamination from the handpiece, and the operator's view is less obstructed.

The cutting can be guided by the bevel of the chisel. Chisels are worthless, of course, without proper malleting. I prefer a mallet with a fiber head, rather than metal, with the head large enough for the assistant to strike the chisel easily. The handle should be light but comfortable in the assistant's hand. The mallet should be heavy enough to give a sharp initial impact but light enough so that follow-through inertia will not jar the patient. A wood carver uses a wooden mallet for the same reason—a chisel seems to cut only at the instant of sharp impact and a heavy mallet only wastes energy and jars the patient unnecessarily without increased efficiency in cutting.

The mallet is held loosely in the assistant's hand. A tendency to hold the handle of the mallet too tightly puts the wrist and arm behind the blow and jolts the patient more than is necessary. Proper malleting does not traumatize the temporomandibular joint nor endanger the integrity of the jaw.

■ *Sectioning of a Tooth*

The amount of bone removed is the least that is consistent with easy removal of the tooth. This is facilitated by the sectioning of many teeth. Sectioning a tooth is like cutting a diamond: one should select a good line of cleavage, usually the anatomic landmarks. Many operators fail in this technic because they do not use sharp chisels. One cannot cut bone with a chisel and expect to use the same chisel to section a tooth. A fresh, sharp chisel should be used each time a tooth is to be sectioned.

The mallet blow in sectioning a tooth is delivered with a light, free flip. If the chisel is sharp and the cutting edge is placed in the proper place, a surprisingly light blow is effective. In splitting longitudinally, the occlusobuccal groove should be used, and in splitting buccolingually the neck or dentinoenamel junction should be selected (Figs. 2 and 3).

In some cases the bone is so hard and eburnated that it is impossible to cut it

with a chisel. In these cases I use a cross-cut tapered fissure bur and change the bur often. Also, in deep horizontal impactions where the crown of the impaction is close to the distal aspect of the second molar, a bur is used to cut, at the neck of the third molar, a space large enough to permit forcing the crown distally and large enough to deliver the tooth from the socket without injuring the second molar.

Each operator has his own technic, based on training and personal experience. Some use a combination of the different technics. I have tried them all for the past thirty years and have had the best success with a mallet and chisel. I believe this technic has its merits if used properly.—*450 Sutter Street.*

The nerve supply to the maxillary incisors

■ *William A. Cook, D.D.S., Detroit*

Despite the fact that human anatomy has been studied diligently since the fifth century B.C., and extensive researches have been carried on by skilled anatomists, the field with which the oral surgeon deals has not been completely explored.

Though it is commonly known that considerable variations from textbook descriptions exist, new anatomy textbooks present the same descriptions of the facial anatomy as their predecessors, with only slight variations in phrasing. This makes almost certain the conclusion that many descriptions have been written without the aid of complete dissections. This certainly is true of the nerve supply of the maxillary teeth. Emphasizing this point is the following question: Has any one ever seen a specimen which shows the innervation of the maxillary teeth, or does any one know where such a specimen can be seen?

It appears that writers have accepted the descriptions of early anatomists, especially Henle,[1] as conclusive and have used those descriptions without checking them with authoritative specimens.

With the advent of block anesthesia, the anatomic structures of the face, especially its nerve supply, assumed prime importance to the oral surgeon. This stimulated considerable interest in dissections of the head and neck, with the result that rather recently important variations from the textbook descriptions have been demonstrated. There are several variations which the oral anesthetist cannot disregard without jeopardizing fully effective results from local anesthetic drugs. Among these are variations in the nerve supply to the maxillary incisors, the common absence of the middle superior alveolar nerve and variations in osseous structures which determine the location of such important points as the mandibular and men-

1. Henle, F. G. J., *Handbuch der systematischen Anatomie des Menschen.* Braunschweig: F. Vieweg & Son, 1871.

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Fig. 1.—Magnified anatomic specimen showing the nasopalatine nerve within the incisive canal and demonstrating its possible course to the incisors: A, nasopalatine nerve; B, branch leaving the nasopalatine nerve and passing upward and forward through the anterior wall of the canal; C and D, fibers passing across the cancellous area and attaching to the heavy strand, E, which traverses the cancellous space and attaches to the apex of the central incisor

tal foramens. These and other variations frequently are noticed by the prudent technician and vigilant clinical observer when his painstaking efforts to block certain nerves meet with failure.

One of the most interesting anatomic problems occurs in the premaxillary region, which contains the maxillary incisors. Until recently it has been generally taught that the maxillary incisors are innervated solely by the anterior superior alveolar nerve. To understand the error in this teaching one need only consider that in cases of bilateral cleft lip and cleft palate it is impossible for the anterior superior alveolar nerve to reach these teeth. It is well known, too, that these teeth have normal pulp canals and normal sensation; therefore, some nerve other than the anterior superior alveolar must supply them with sensory fibers.

■ *Possible Role of the Nasopalatine Nerve*

It has not been proved that the nasopalatine nerve plays a part in this innervation, but there is well supported evidence for the theory that the nasopalatine nerve alone supplies sensory fibers to the maxillary incisors in persons with bilateral cleft lip and cleft palate and contributes to their innervation in normal persons.

During the last thirty years, many attempts have been made to dissect out and thereby identify the nerves supplying the maxillary incisors, but apparently this has not been accomplished. Several highly skilled dissectors among the teaching anatomists have tried to solve the problem, but none has succeeded.[2]

It is not strange, however, that a dissection showing the nerves supplying the maxillary incisors has not been achieved, because in the cancellous space above these teeth there is a highly complex entanglement of nerve fibers, blood vessels and strands of fascia (Fig. 1, *E*), which can be disentangled only with great skill or a lot of luck.

2. A few years ago, a dissector in a refresher course reported that he had completed a dissection showing a branch of the nasopalatine nerve running to the central incisor. No records were made, and the specimen, which, if authentic, would have been worthy of a place in the Smithsonian Institution, was destroyed.

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Fig. 2.—Anatomic specimen showing the nasopalatine nerve in its course across the nasal septum

To support the belief that the nasopalatine nerve does innervate the maxillary incisors, the following facts and theories are submitted for consideration:

The nasopalatine nerve (Figs. 2 and 3), like the anterior, middle and posterior palatine nerves, is a branch of the two or three sphenopalatine nerves (Figs. 4 and 5), which are branches of the maxillary (the second division of the trigeminal) nerve.

The sphenopalatine nerves are only a few millimeters long. They branch from the maxillary nerve in the sphenomaxillary fossa and pass directly downward to the sphenopalatine ganglion (Figs. 3 and 4), which they enter and within which they branch into the nasopalatine and anterior, middle and posterior palatine nerves. However, these nerves do not become a part of the ganglion, but pass directly through the ganglion capsule (Fig. 4) to its inferior border, from which they emerge and continue to their various destinations.

The nasopalatine nerve leaves the lower anterior portion of the capsule, passes medially through the sphenopalatine foramen (located at the junction of the palatal, maxillary and sphenoid bones) into the posterior nasal fossa. Continuing medially, it traverses the roof of the fossa to the nasal septum, in which it passes downward and forward to the anterior part of the floor of the nasal fossa. There it enters the incisive canal (Figs. 1 and 3), through which it descends to the palate to supply the tissues in the incisive region. In its entire course it gives branches to adjacent tissues.

■ *Reasons for Belief*

The belief that the nasopalatine nerve sends fibers to the maxillary incisors is based on several factors.

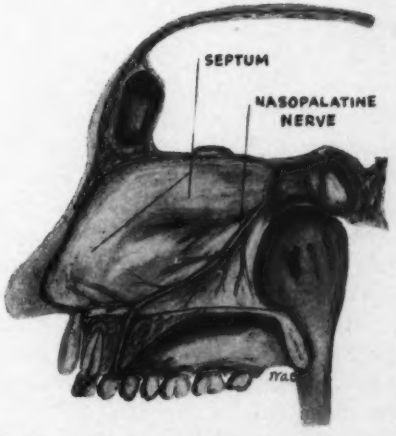

Fig. 3.—Conception of the nasopalatine nerve in the nasal septum and its distribution

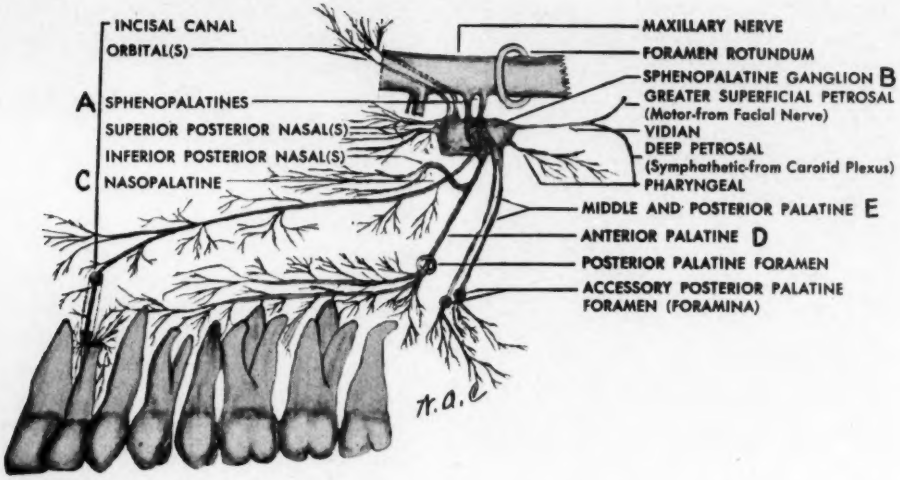

Fig. 4.—Conception of, A, the sphenopalatine nerves; B, sphenopalatine ganglion; C, nasopalatine nerve; D, anterior palatine nerve; E, middle and posterior palatine nerves

1. No one, so far as I have been able to learn, has been able consistently to anesthetize the upper incisors by anesthetizing the anterior superior alveolar nerve alone.

2. On many occasions when an attempt to anesthetize the upper incisors with a precise block of the anterior superior alveolar nerve has failed, an injection high in the incisive canal has produced the desired result. Since the nasopalatine nerve is the main nerve in the incisive canal (the others probably are branches from the anterior superior alveolar nerve), it is reasonable to assume that the nasopalatine nerve sends fibers to the incisors, and that anesthesia of these fibers desensitizes the teeth.

3. The upper incisors often are at least partly desensitized when the rhinologist anesthetizes the nasal septum with cocaine packs. The nasopalatine nerve is the only nerve anesthetized by this means which could innervate the teeth in the premaxilla. Therefore, the fact that they are desensitized by this procedure makes it logical to assume that at least part of their nerve supply is from the nasopalatine nerve. Of course, this assumes that the anesthetic solution applied to the nasal mucosa does not penetrate the soft and osseous tissues overlying the anterior superior alveolar nerve in its course within the cancellous bone above the incisors. Such penetration seems impossible.

4. Considered from an embryologic standpoint, the nasopalatine nerve is the logical source of innervation to the maxillary incisors. That conclusion can be grasped immediately when the develop-

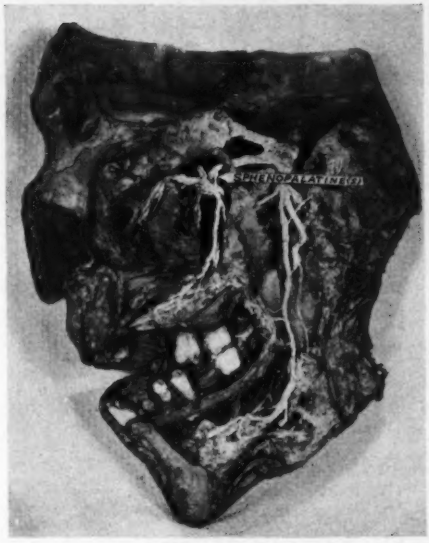

Fig. 5.—Anatomic specimen with arrow indicating the sphenopalatine nerve (or nerves) passing from the maxillary nerve to the sphenopalatine ganglion

ment of the oral cavity is considered. Piersol[3] wrote:

> The upper and lower boundaries of the primitive oral cavity and the differentiation of the nasal region proceed from three masses, the two lateral paired maxillary processes of the first visceral arches and the mesial unpaired frontonasal process, which descends as a conspicuous projection from the ventral surface of the anterior part of the head. The maxillary processes grow toward the midline and, in conjunction with the descending frontonasal projection, form the lateral and superior boundaries of the primitive oral cavity.

The frontonasal process (Fig. 6) is the origin of the nasal septum, including the septal cartilage, the perpendicular plate of the ethmoid bone and the vomer. The front of the nose and the premaxilla, in which the incisors develop, also originate in the frontonasal process.

The nasopalatine nerve develops on the lateral surface of the frontonasal process. Therefore, the nasopalatine nerve and the upper incisors develop together and continue in close relation. These facts suggest that the nasopalatine nerve naturally innervates the teeth in the premaxillary bone.

The maxillary processes and the frontonasal process are entirely separate until about the seventh week of fetal life, when they usually unite. Failure to unite produces cleft lip and cleft palate. The clefts occur in the line between the lateral incisors and the cuspids and extend posteromedially to what normally would be the incisive canal. Hence a triangular segment of bone, the premaxilla, is left

3. Piersol, G. A., *Human Anatomy, Including Structure and Development and Practical Considerations*, ed. 9. Philadelphia: J. B. Lippincott Company, 1930, pp. 1230-1240.

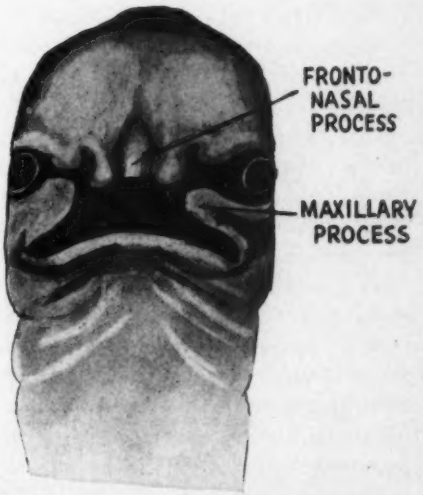

Fig. 6.—Head of an embryo of about thirty-three days, demonstrating the frontonasal and maxillary processes. (From Piersol.[3])

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Fig. 7.—Child with bilateral cleft lip and cleft palate, showing the premaxilla attached only to the vomer

entirely separated from the maxillas and hangs as a projection at the end of the vomer (Fig. 7). It would be impossible, therefore, in a person with bilateral cleft lip and cleft palate, for the anterior superior alveolar nerve to supply the incisors.

5. Supporting the theory further is the fact that the superior alveolar and nasopalatine nerves are morphologically identical. Both are branches of the maxillary division of the fifth cranial nerve. They have identical fiber origin and construction, and perform the identical functions of receiving sensory impulses and transmitting them to their common center, the trigeminal reception nucleus.

6. It has been stated by Henle that the nasopalatine nerve, before entering the incisive canal, gives an anastomosing branch to the anterior superior alveolar nerve and that in the incisive canal the two nerves form a plexus. But Henle described the distribution of the nerves, in the plexus, to the anterior portion of the premaxillary mucosa and said nothing about the innervation of the teeth.

7. That nerve filaments from within the incisive canal do pass through the anterior wall and into the cancellous region above the incisors is demonstrated in Figure 1. In this specimen, a branch from the nasopalatine nerve, in the incisive canal, is seen leaving the anterior surface of the nerve trunk. It passes upward and forward through the anterior wall of the canal and enters the cancellous region above the incisors, then attaches to the heavy strand (Fig. 1, *E*) which travels downward to the apex of the central incisor. Owing to the complexity of the strand, I was unable to work through it. However, the dissection demonstrated a course by which the maxillary incisors could be innervated by the nasopalatine nerve.

■ *Comments*

From the evidence here submitted, it is reasonable to assume that the nasopalatine nerve is the sole nerve supplying the maxillary incisors in persons with bilateral cleft lip and cleft palate, and it is equally reasonable to assume that the nasopalatine nerve sends fibers to these teeth in normal persons.

This problem has been presented to anatomists, neuroanatomists and embryologists, who expressed general agreement that the theory of the nasopalatine nerve sending fibers to the maxillary incisors is reasonable and probable.—*1850 David Whitney Building.*

Tumor of Küttner

■ *Michael N. Papoulacos, M.D., Athens, Greece*

The so-called tumor of Küttner, or inflammatory tumor of the salivary glands, is a lesion of exceptional interest from the point of view of clinical and pathologic diagnosis. Although described by Blair[1] and emphasized by Swinton and Warren,[2] it seems not to have attracted the attention of surgeons and pathologists.

First described as a clinical entity by Küttner[3] in 1896, it is a benign lesion of the salivary glands presented as a tumor and usually diagnosed as a malignant neoplasm. The pathologic report of "chronic inflammatory process" will surprise the surgeon. On the other hand, its microscopic diagnosis requires special knowledge of such benign tumors.

The following report of 2 cases is presented in the hope that it will call attention to the frequency of this tumor, and other cases will be reported.

■ *Report of Cases*

CASE 1—A man 32 years of age stated that for about a year and a half he had had a hard, indolent swelling in the right submaxillary region. It grew gradually to the size of a walnut. The patient complained of a sensation of pressure on swallowing, but had not observed any excessive salivation or dryness of the mouth. His past history was generally noncontributory.

Examination showed a tumor the size of a large walnut in the area of the right submaxillary gland. The tumor was smooth, hard, indolent and immovable. It was palpable from the buccal cavity, but did not protrude into it. The mucosa over the tumor was normal but immovable. No lymph nodes were palpable. Roentgen study did not show stone. The Wassermann reaction was negative.

The tumor was removed on June 9, 1937. During the operation adhesions to the oral mucosa were found.

The pathologist reported chronic productive diffuse inflammation, which had developed into sclerosis of the salivary gland. Elements of neither neoplasm nor a specific inflammatory process were seen. Giemsa and Gram staining showed no microbes, and no actinomycetes were seen. Culture of fluid extracted from the gland gave negative results.

Recovery was uneventful. Eleven years after the operation there had been no recurrence.

CASE 2—A man 53 years old said that for three years he had noticed a tumor of the left submaxillary region.

Examination showed a hard, lobulated, indolent and immovable tumor the size of a large walnut. At first a diagnosis of sialolithiasis was made. Roentgen examination showed no shadow of stone. The Wassermann reaction was negative.

From the Department of Surgery (Dr. M. Makkas, Director) of the Greek Red Cross Hospital, Athens, Greece.

1. Blair, V. P., *Surgery and Diseases of the Mouth and Jaws*, ed. 3. St. Louis: C. V. Mosby Co., 1917, pp. 498-499.

2. Swinton, N. W., and Warren, S., Salivary Gland Tumors. *Surg., Gynec. & Obst.* 67:424 (Oct.) 1938.

3. Küttner, Hermann, Ueber entzündliche Tumoren der Submaxillarspeicheldrüse. *Beitr. z. klin. Chir.* 15:815, 1895-1896.

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The patient was operated on May 12, 1947, under a tentative diagnosis of malignant tumor of the left submaxillary region, with a second consideration that, if the growth proved to be in the submaxillary gland, it could be a tumor of Küttner. The latter supposition was based on the earlier case. At operation the tumor was found to involve the submaxillary gland. It was firmly attached to the surrounding tissues.

Macroscopically the growth resembled a malignant neoplasm, but microscopic examination showed no malignant elements, but benign chronic productive sialadenitis in the category of the so-called inflammatory tumor of Küttner. No elements of specific inflammation were present, and neither microbes nor actinomycetes were seen in sections and cultures.

Recovery was uneventful.

■ *Symptoms*

The tumor of Küttner appears as a hard swelling in any of the salivary glands, usually the submaxillary gland. It may grow gradually and painlessly for years, at times reaching the size of a large apple. Frequently there is a sensation of heaviness at the site of the tumor during mastication, swallowing, talking or yawning. The general health remains good.

The tumor is hard and smooth or lobulated. It is characteristically immovable on account of solid adhesions to the surrounding tissues, especially to the buccal mucosa. Such adhesions are observed even when the tumor is of recent origin. In 10 per cent of cases one to three acute inflammatory attacks of short duration intervene during its slow development. The opening of the salivary duct remains normal in any case. The corresponding lymph nodes do not swell, though Küttner[4] described infiltration in most of his cases. No sialorrhea is present. The blood picture does not change.

■ *Pathologic Picture*

The term "tumor" is applied to this condition because a mass is formed in an organ surrounded by a connective tissue capsule. On section the specimen creaks under the knife. The cut surface shows a lobular structure resembling that of a salivary gland.

According to Lang[5] and Kaufmann,[6] the principal characteristics of the histologic picture are: the development of abundant fibrous connective tissue, separating the acini and at places enclosing lobule-like sections of the glandular parenchyma of various sizes; the presence of inflammatory infiltrations consisting mainly of polymorphonuclear leukocytes and plasma cells; the destruction of glandular parenchyma. The lumens of the ducts, because of thickening of the walls and the microcellular infiltration, take the appearance of torn channels. The presence of sand or minute stones (in 9 per cent of cases) should be considered secondary.

The process has nothing to do with syphilis, tuberculosis or Mikulicz's disease.

■ *Diagnosis*

Clinical differentiation between Küttner's tumor and malignant neoplasm is

4. Küttner, Hermann, Die Chirurgie der Speicheldrüsen, in Bergmann, E. von, *Handbuch der praktischen Chirurgie*, ed. 6. Stuttgart: F. Enke, 1926, vol. 1.

5. Lang, F. J., Pathologische Anatomie der grossen Kopfspeicheldrüsen, in Henke, F., and Lubarsch, O., *Handbuch der speziellen pathologischen Anatomie und Histologie*. Berlin: Julius Springer, 1929, vol. 5, part 2.

6. Kaufmann, Eduard, *Lehrbuch der speziellen pathologischen Anatomie für Studierende und Aerzte*. Berlin: W. de Gruyter & Co., 1931, vol. 1.

difficult, at times impossible. Plain roentgenograms and sialograms may help in diagnosis. Diagnosis always is established by histologic examination. Biopsy may be of great aid, especially when the parotid gland is involved. It must be emphasized that special attention is needed for histologic examination. At times even microscopic differentiation between this benign process and a malignant growth can be difficult.

■ *Incidence*

Tumor of Küttner is believed to be rare, although Küttner[7] observed 5 cases in two years after his first communication, and later drew the attention of surgeons many times to the condition. Heineke[8] in 1913 had collected 18 cases, and Küttner[4] by 1926 had collected 27 cases. The total number reported now has reached 51.[9] The cases reported bring the total to 53.

It should be emphasized that physicians who keep the condition in mind will find it not so infrequent as it is generally believed to be.

This tumor may occur in any of the three salivary glands. In 35 cases the submaxillary gland was involved; in 13, the parotid; in 2, the sublingual, and in 3, both the submaxillary and the sublingual.

As a rule, the swelling is unilateral. Bilateral localization was mentioned in 4 cases, 2 of submaxillary and 2 of parotid involvement. Predilection for the right or the left side cannot be determined, for the side of involvement has not been stated in all reports. The disease usually occurs in middle age, although a girl of 3 years and a man of 79 have been affected. The disease attacks men (30 cases) more often than women (18 cases). In 5 reports sex was not mentioned.

Profession, trade and use of tobacco or alcohol do not seem to influence the process.

■ *Cause*

Most authors have expressed the opinion that the condition probably is due to entrance of microorganisms through the duct. Microbiologic search was made during the pathologic examination. Some investigators (Kroiss,[10] Städtler[11]) found scattered streptococci or Gram-positive or Gram-negative cocci. The others were unable to demonstrate any microorganism, nor was I. Culture was done in only 5 cases besides the 2 reported here. Results were negative in 6,[12,13] but McKenna and Davis[14] found pneumococcus.

Söderlund[15] reported the presence of actinomycetes in some cases, and expressed the belief that the lesions he observed were inflammatory tumors, in the sense used by Küttner. But fistula was present in these cases. This favors the diagnosis of actinomycosis. As a matter of fact, fistula never has been observed in

7. Küttner, Hermann, Ueber entzündliche Tumoren der Submaxillarspeicheldrüse. *Arch. f. klin. Chir.* 57:873, 1898.

8. Heineke, Hermann, *Verletzungen und chirurgische Krankheiten der Speicheldrüsen.* Stuttgart: F. Enke, 1913.

9. Papoulacos, M. N., *Inflammatory Tumor of the Salivary Glands, or Tumor of Küttner.* Doctors' Thesis, University of Athens, 1944.

10. Kroiss, F., Ueber die chronische Entzündung der Mundspeicheldrüse und ihrer Ausführungsgänge. *Beitr. z. klin. Chir.* 47:470, 1905.

11. Städtler, Ueber chronisch interstitielle unspezifische Speicheldrüsenentzündung. Dissertation, Erlangen, 1935.

12. Abadie, Scalone and Cevario, cited by Papoulacos.[9]

13. Tait, D., Fibrous Atrophy of the Parotid Gland, with Especial Reference to the Treatment of the Salivary Fistula: An Experimental and Clinical Study. *Surg., Gynec. & Obst.* 14:456, 1912.

14. McKenna, C. H., and Davis, D. J., Chronic Suppurative Parotitis Caused by the Streptococcus Mucosus Capsulatus: Report of a Case. *J.A.M.A.* 54:47, 1910.

15. Söderlund, G., Die Speichelsteinkrankheit ("Sialolithiasis") und ihr Verhalten zu der primären und duktogenen Speicheldrüsenaktinomykose. *Acta chir. Scandinav.* (supp. no. 9) 63:1, 1927.

typical Küttner tumor. However, Söderlund maintained that primary actinomycosis, salivary lithiasis and tumor of Küttner have the same source. This conclusion certainly is not justifiable. Later on, other authors, for example Ganner,[16] noticed the presence of rare actinomycetes in the involved glands characterized as Küttner tumor. But in Ganner's case, after the removal of the tumor, a fistula remained. This discharged pus, in which actinomycetes were observed. Cases in which these findings occur should be classified as instances of actinomycosis of the salivary gland and not of Küttner's tumor. It should be noted here that in the cases in which actinomycosis was found, it was discovered in a few foci only. In Ganner's case 1,000 sections were necessary to demonstrate the presence of actinomycetes. Numerous sections from several parts of the tumors were examined in the cases reported here. Long search showed neither actinomycetes nor cocci.

How can the tumor of Küttner be caused by common pyogenic microorganisms, such as pneumococci, streptococci and actinomycetes, when after special search they are found so rarely? These agents cause tissue softening, necrosis and purulence, but they abound in the buccal cavity. If they were the cause, they would appear more consistently in microscopic sections. It is well known that these microorganisms multiply so profusely in the tissues which they attack that their detection is easy, not only in cultures, but even in plain microscopic slides.

I believe, therefore, that Küttner's tumor probably represents a pathologic process *sui generis* of yet unknown cause. If its histologic picture forces its acceptance as an inflammatory process, the term "inflammatory" must be used in the broadest sense, and no effort should be made to relate it to the common microorganisms which produce pyogenic inflammation.

The finding of a few microorganisms in certain cases is not difficult to explain. The penetration of organisms from the mouth into the salivary glands occurs readily whenever the flow of saliva is not normal. If during this passage the organisms find in the duct sinuses stagnant secretion containing desquamated epithelium, they may multiply there, yet have no etiologic relation to the preexisting process. Actinomycetes or other pyogenic organisms might settle in a preexisting tumor of Küttner because of unusually favorable local circumstances or of great toxicity, and might produce secondary actinomycosis or secondary suppurative inflammation.

■ *Treatment*

The treatment which should be recommended is removal of the tumor. This is definite when the submaxillary or the sublingual gland is involved. For parotid disease, however, caution is advisable. Physical therapy may be used.

When the diagnosis is doubtful, frozen sections should be made during the operation, and treatment determined according to the observations, to avoid the danger of missing a definitely malignant tumor.

Total parotidectomy should be performed only when absolutely necessary. Experience has taught that after partial

16. Ganner, H., Ein Fall von Aktinomykose der Unterkieferspeicheldrüse, zugleich ein Beitrag zur Frage der Aetiologie der sogenannten küttnerschen Speicheldrüsentumoren. *Arch f. klin. Chir.* 155:495, 1929.

excision of the tumor, the disease may recede in the remaining part of the gland.

No recurrence ever has been reported.

■ *Summary*

1. The so-called tumor of Küttner, or inflammatory tumor of the salivary glands, constitutes a separate clinical category of chronic, nonspecific productive inflammation appearing as a tumor.

2. Fifty-three cases have been reported (including 2 reported here). With greater appreciation of the frequency of the lesion, probably more cases will be observed in the future.

3. Diagnosis is of particular interest, for the growth as a rule is considered malignant.

4. Usually the tumor is unilateral, and in any of the three salivary glands. It occurs mainly in middle age, and more often in men than in women.

5. Treatment is by excision of the tumor.

6. The process probably is peculiar only to the disease, though its exact nature still escapes medical knowledge. In any event, it is not related to the common microorganisms which produce pyogenic inflammation.

■ Graduate Course Offered by the University of Illinois

A two-year, full-time graduate course in the basic sciences, with limited clinical experience, is being offered by the Graduate School of the University of Illinois, through the department of oral and maxillofacial surgery.

The course will carry credit toward a Master of Science degree. Applications now are being accepted for September 1949.

Further information can be secured from the Department of Oral and Maxillofacial Surgery, College of Dentistry, University of Illinois, 808 S. Wood St., Chicago 12, Ill.

■ Residencies in Maxillofacial and Oral Surgery

The Department of Oral Surgery at the University of Michigan offers residencies in the fields of maxillofacial and oral surgery.

The fellowship in oral surgery is open to two men each year. Appointments begin in February and June and continue for three years, leading to the degree of Master of Science. Graduates of class A dental schools are eligible.

The residency in maxillofacial surgery is for a two year period. One appointment is made each year. Applicants must have dental and medical degrees, and at least one year of rotating internship.

UMI

Reticulum cell sarcoma of the malar zygomatic region: *report of case*

■ *Arthur R. Dewey, D.D.S., Ventura, Calif.*

Sarcoma has been defined as a tumor arising from connective tissue and retaining most of the general characteristics of connective tissue, but it is endowed with the power of invading and actively destroying adjacent structures and forming colonies of its own tissue in distant organs. In this respect sarcomas are typically malignant tumors.

In these tumors, which tend to metastasize through the blood vessels rather than through the lymphatic vessels, there is usually one kind of cell which predominates, and the name of the tumor is derived from this variety of cell.

Thoma[1] states that the clinical history often associates trauma with the appearance of the tumor. Such observations usually lack trustworthiness since the trauma might have occurred after the onset of the disease. An existing sarcoma is often traumatized, especially in the mouth, which leads to its more rapid development and discovery.

A case is presented in which a swelling, later diagnosed as a reticulum cell sarcoma, was noted in the adjacent structures shortly after the difficult removal of an impacted maxillary third molar tooth.

HISTORY—A 20 year old white school girl was seen June 20, 1946. She complained of a lump, about the size of an olive, which had appeared near the right lateral orbital margin. She stated that about three months previously a severely impacted right maxillary third molar tooth was removed. The operation required approximately an hour and a half, and the postoperative course was long and stormy.

A few weeks after the extraction the lump appeared, and somewhat later a mass appeared in the right side of the throat, displacing the right tonsil medially. Recently she developed a diplopia and a paresthesia of the right face.

She was examined by an optometrist, but he was unable to correct the diplopia. Her family physician referred her to this office for possible diagnosis and treatment.

EXAMINATION—Examination revealed a well developed, well nourished young white girl with a firm, nonpainful, nonfluctuant, deep-seated, adherent mass, about 1.5 cm. in diameter, located near the lateral orbital margin over the body of the zygoma on the right.

A paresthesia of the structures supplied by the fifth cranial nerve could be demonstrated, and the patient complained of double vision in the right eye.

Oral examination revealed a normal appearing and well kept mouth. The right tonsil extended into and partially obstructed the oral pharynx. The remainder of the physical examination revealed nothing.

Roentgenographic examination of the region, consisting of a stereoscopic Waters

1. Thoma, K. H., *Oral Pathology*, ed. 2. St. Louis: C. V. Mosby Company, 1941.

examination of the facial bones, disclosed a destructive process of osteolytic character involving the body and part of the arch of the right zygoma. Definite outline of the bone was no longer possible because of the extensive demineralization of the osseous structure. There was no evidence of any extension into the orbit or the maxillary sinus.

In view of the clinical and roentgenographic observations, it was thought imperative that microscopic examination of the involved tissues be made.

OPERATION—The patient was hospitalized, and on July 2, an operation was performed to obtain tissue from the lesion for histologic and pathologic study.

A premedication of 1/6 grain morphine sulfate and 1/150 grain atropine sulfate was administered one hour before the operation. Under intravenous thiopental sodium anesthesia, an intraoral incision was made in the right maxillary buccal fold. The tissues were divided by blunt dissection until the region surrounding the zygomatic bone was accessible. No tumor tissue could be identified or located by this approach. The incision was closed with interrupted catgut sutures. A second sterile setup was obtained and the tissues overlying the tumor were prepared for an external approach to the lesion.

A horizontal incision of approximately 2 cm. was made just above the superior border of the zygomatic arch and directly overlying the swollen region. The tissues were carefully separated by blunt dissection to avoid injury to any of the facial nerve fibers. The region of the zygomatic arch was exposed, adjacent to which diffuse reddish blue neoplastic tissue was found. A specimen was taken and it was sent to the laboratory for analysis. The incision was closed without a drain, and a dry dressing was applied.

MICROSCOPIC EXAMINATION—The neoplastic tissue revealed masses and cords of tumor cells in which there were fragments of striated muscle tissue in various stages of degeneration. The tumor cells were small, round to spindle shaped, with frequent mitosis and they exhibited pleomorphic and hyperchromatic characteristics. There was no attempt at formation of any definite tissue or glandular pattern.

DIAGNOSIS—The diagnosis was sarcoma of undifferentiated type. The final classification rested with the results of special staining procedures. The possibilities were: reticulum cell sarcoma; undifferentiated round cell sarcoma; fibrosarcoma of undifferentiated or embryonal types.

As a result of special staining, a final diagnosis of reticulum cell sarcoma was made.

COURSE AND TREATMENT—After consultation with the surgical and roentgenographic departments, it was decided that because of the position and extent of the lesion, a radical surgical resection would accomplish nothing except extreme mutilation of the patient.

Since the reticulum cell type sarcoma responds most effectively to roentgen radiation, it was decided that deep roentgen therapy was the logical treatment.

Between July 11 and July 30, 1946, deep roentgen therapy was administered to the anteroposterior and lateral ports, 10 by 10 cm. in size. The factors were 200 kv., peak, ½ copper, 1 aluminum filtration, 50 cm. distance, half-value layer 1 mm. copper. The patient was given a total of 1,350 roentgen units to each of the two fields.

Roentgenographic examination of the chest for lung metastasis was negative.

After roentgen therapy, the local tumor regressed completely, and displacement of the right tonsil was no longer noted. The patient continued to be without symptoms until December 1946, at which time blurring of the right eye developed. Roentgenographic examination at this time showed considerable remineralization of bone in the involved portion of the zygoma, and roentgenographic examination of the chest remained negative. The patient was referred for an eye examination.

On February 12, 1947, the patient complained of deafness of the right ear, headaches, weakness and dizziness. Roentgenograms failed to show any recurrence of bony destruction. However, the entire right zygomatic bone was definitely less mineralized

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than the one on the left, and roentgen treatment was resumed with the same factors. Four treatments, 200 roentgen units each, were given through the right temporal region.

The patient's parents desired further consultation, and were referred to the Mayo Clinic at Rochester, Minnesota. Fred Z. Havens, M.D., of the Mayo Clinic, reviewed the history and examined the patient. His conclusion was that the tumor was in an inoperable position, that radiation therapy was the indicated treatment and that the prognosis was extremely grave with little hope.

The patient returned to California, and between May 20 and 28, 1,200 roentgen units were given to the upper temporal region, using the same factors as were used in previous treatments.

By this time the lesion appeared to have become radioresistant and was progressing unchecked by further roentgen therapy. Palliative treatment was indicated.

On September 17, the patient suddenly died while at home. The parents refused permission for a postmortem examination.

DISCUSSION—This sarcoma, discovered a short time after the difficult surgical removal of an impacted tooth, again raises the question of what part trauma plays in the etiology of malignant lesions. Was the formation of the neoplastic disease merely coincidental with the trauma, did trauma act as the exciting factor of an already existing malignant lesion and lead to a more rapid development and discovery, or did the trauma, associated with the removal of a tooth, produce some unknown change in the adjacent cells which caused them to undergo a malignant degeneration?

No doubt the incidence of trauma plays a very important part in the disease.

An early diagnosis is of prime importance. Radical excision of the primary tumor in the earlier stages is the only hope for a cure of the condition. These growths are very cellular, and after surgical intervention there is a tendency towards rapid local recurrence unless all of the involved tissue is removed. The chief danger lies in the rapid and widespread dissemination of the growth to distant organs.

It is characteristic of sarcoma to diminish rapidly in size with radiation treatments, but this does not mean the process is finished, as recurrence is the rule with increased rapidity of growth. Metastasis is usually to the lungs.

The possibility of radical resection was considered in the reported case. However, in staff consultation, it was decided to resort merely to radiation therapy. This decision was later supported when the girl was examined, and the history was reviewed at the Mayo Clinic.

The decision against radical surgical excision of the growth was made (1) because of the already present evidence of dissemination of the tumor to the tissues of the throat, as noted by the displacement of the tonsil into the oral pharynx, (2) because of the neurologic involvement, as evidenced by the trigeminal and optic nerve disturbances and (3) because of the location of the tumor which would have necessitated severe and extreme mutilation of a young girl.

The prognosis in this case was very poor. A small per cent may be expected to survive three to five years. In this case with the tumor radioresistant and progressing unchecked the time limit was reduced.

CONCLUSIONS—A growth, later diagnosed as a reticulum cell sarcoma, was noted in the adjacent structures after the difficult removal of an impacted maxillary third molar tooth.

Because of the location and extent of the growth, it was decided, upon consultation, that radical excision was contraindicated and radiation therapy was used.

The lesion initially reacted favorably to the radiation but later became radioresistant and progressed unchecked.

The patient died suddenly four months after the final roentgen treatment.—*2427 E. Main Street.*

Spindle cell sarcoma of the hard palate: *report of case*

■ *Philip M. Northrop, D.D.S., M.S., Ann Arbor, Mich.*

Early in 1943 a white woman, aged 56, presented herself because of swelling in the palate.

HISTORY—The patient had first noticed a small mass beneath her maxillary denture eight months before. The lesion had enlarged gradually. The lesion had been asymptomatic except that it became impossible to keep the denture in place. When she first noticed the tumescence she consulted a dentist who advised observation. With the continued increase in the size of the mass she became alarmed and sought other counsel.

EXAMINATION—The patient's general physical condition was not good. For years she had been under treatment for hyperthyroidism and hypertension. A complete physical examination verified these complaints, showing blood pressure 240 systolic and 110 diastolic, with definite cardiac changes.

The patient was extremely apprehensive and nervous. She was edentulous, and a soft fluctuant pedunculated mass approximately 5 by 3 cm. was present on the left side of the hard palate (Fig. 1).

PATHOLOGIC STUDY—A section of the mass removed for microscopic examination showed spindle cell sarcoma, probably following malignant transformation of a mixed salivary gland tumor (Fig. 2). Complete and radical removal of the tumor was advised.

OPERATION—On May 26, 1943, after heavy premedication, nitrous oxide was administered by means of endotracheal intubation. With a cutting current, the soft tissue was incised deeply to the bone and widely around the mass. The incision extended medially to the area of the posterior palatine foramen, posteriorly well into the soft palate and anteriorly into the vestibule and along the mucobuccal fold to the tuberosity of the maxilla. With a periosteal elevater the soft tissue within the incised area was elevated from the bone. The mass was seen to have encroached on the floor of the nose and extended well into the maxillary sinus. Because of the nature of the tumor, the only possibility of recovery lay in its radical removal. Therefore, all the left side of the maxilla up to the infraorbital area, the molar ridge, the floor of the nose over to the right posterior part of the palatine area and the left pterygoid plates were removed. Some bleeding was encountered, but its control was not difficult. The entire operative defect was packed with bismuth tribromophenate gauze.

From the department of oral surgery, University of Michigan, Ann Arbor, Mich.

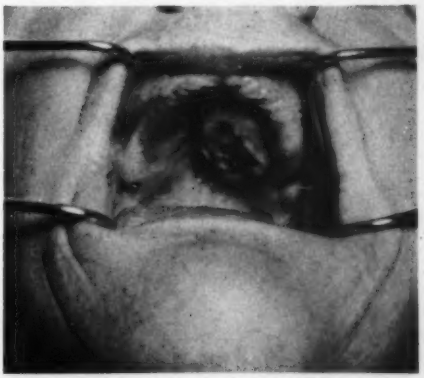

Fig. 1.—Spindle cell sarcoma of the palate (before operation)

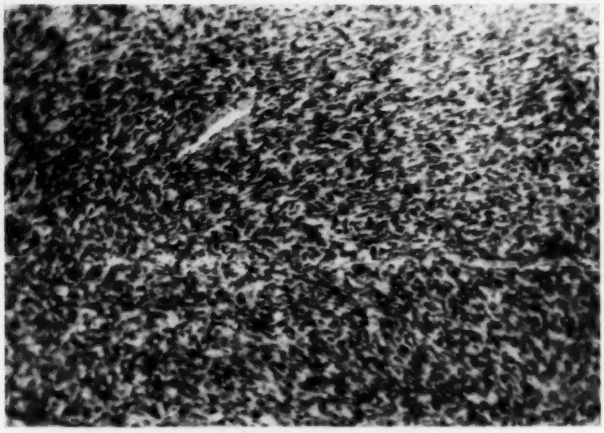

Fig. 2.—Neoplasm composed of small spindle cells which are characteristic of a spindle cell sarcoma. Glandular elements are also present in other areas of the tissue examined

POSTOPERATIVE TREATMENT—The patient made an unusually good recovery. The dressing was removed in two days. Although the exposed operative site was large, the patient was not unusually distressed, and complained only of soreness. She was discharged one week after operation, June 3, 1943.

PROGRESS—The patient was seen at frequent intervals after her discharge from the hospital. It was interesting to note the rapid closure of the operative defect. One year after the operation, an opening of approximately 2 by 1 cm. remained. This was closed by means of a small prosthetic appliance. After the first year the patient was seen at six month periods. When seen in March 1948, she was examined with great care and there was no evidence of recurrence of the lesion. On April 18, 1948, the patient died as a result of a cardiac accident.

COMMENTS—This case illustrates the fact that mixed salivary gland tumors may undergo malignant change and stresses the importance of early recognition and removal of any tumor in the oral cavity. In this case a dentist had seen the tumor and continued to advise observation. Any mass which has been present in the oral cavity, or in any location, for several weeks should be looked on with concern. A specimen or the whole tumor should be removed, and the tissue should be examined microscopically.

Probably the most dangerous tendency for the surgeon is toward conservatism in the removal of neoplasms, especially of borderline neoplasms. Excision should be radical, since further surgical intervention in the event of recurrence often is of no avail.

■ WESTERN PENNSYLVANIA SOCIETY OF ORAL SURGEONS

Nineteen district oral surgeons met on December 2, 1948, and organized the Western Pennsylvania Society of Oral Surgeons. A constitution was adopted and officers were elected. H. Kingsley Elder was elected president; Reed P. Rose, vice president; Donald E. Wagner, secretary-treasurer.

UMI

Chondrosarcoma of the mandible:
report of case

■ *M. D. Gruber, D.D.S., M.S., Iowa City, Iowa*

This case of chondrosarcoma of the mandible is presented because of its rarity. Padgett[1] states, "it is estimated that one bone sarcoma is present in 75,000 to 100,000 people and that possibly three per cent of the whole group occur in the jaw bone."

HISTORY—Early in 1948, the patient, a 29 year old man, was struck on the right angle of the mandible by a piece of metal spring. In November, the patient complained of soreness in the region distal to the lower right third molar because the upper second molar impinged upon the soft tissue (Fig. 1). The patient's dentist extracted the upper second molar. Two weeks later the soft tissue was no longer tender, but a hard mass was observed in the region distal and lingual to the lower right third molar. The patient was referred to the Oral Surgery Department, State University of Iowa Hospital, for treatment December 16.

EXAMINATION—Oral examination showed upper and lower partial dentures. The mouth hygiene was good. In the region lingual and distal to the lower right third molar was a hard painless mass about 1.5 cm. in diameter. On the gingival tissue lingual to the the lower right third molar was a soft tissue mass having the appearance of a granuloma. The patient complained of partial numbness of the right lower lip of six months' duration.

Fig. 1.—Oral appearance of tumor

Fig. 2.—Roentgenogram showing growth posterior to lower right third molar with characteristics of osteoma

Specimens of bone and soft tissue were taken on December 17 for biopsy.

Roentgenographic examination revealed a tumor of bony density posterior to the lower right third molar tooth, projecting upward approximately 1 cm., and with a diameter of 1.5 cm. (Fig. 2). It was tentatively diagnosed as osteoma of the right side of the mandible.

On January 7, 1949, the biopsy report was received. At this time the growth had

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1. Padgett W. C., *Surgical Diseases of the Mouth and Jaws*. Philadelphia: W. B. Saunders Company, 1938.

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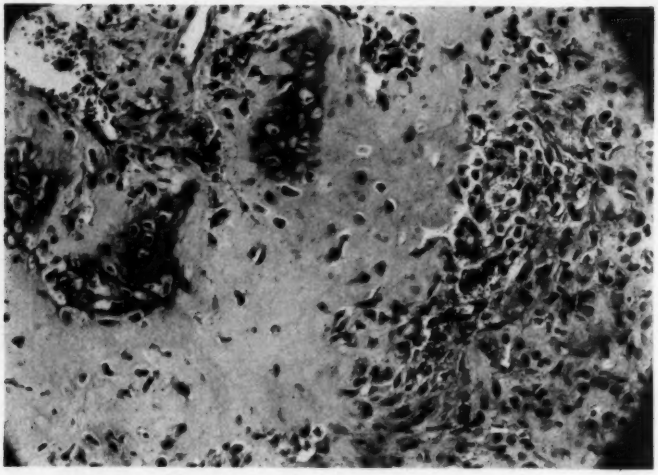

Fig. 3.—Section of bone fragment revealed poorly formed cartilage and osteoid tissue, irregularly calcified

increased in size to about 2 cm. The biopsy report revealed sections of the soft tissue to be skeletal muscle infiltrated by inflammatory cells. There was no evidence of a malignant growth.

Sections of the bone fragment (Fig. 3) showed it to be composed of poorly formed cartilage and osteoid tissue which was irregularly calcified. The lesion appeared well encapsulated by a hyalinized fibrous tissue wall in which were numerous vascular channels and acute and chronic inflammatory cells. Scattered throughout the osteoid cartilaginous tissue were large and small islands of cells which resembled chondroblasts and osteoblasts. These cells were fairly uniform in size, and they were large, with hyperchromic nuclei and with abundant cytoplasm. In areas the nuclei were irregular in outline, with many mitotic figures. Many of the cells were vacuolated. The anaplasia in the more cellular areas, together with the mitotic activity indicated a malignant growth.

The abundance of cartilage formation, together with the characteristics given in the foregoing description, substantiated a diagnosis of chondrosarcoma of the right mandible.

The patient's physical examination showed nothing unusual. There were no palpable nodes. Serum phosphorous, phosphatase and calcium were within normal limits. The Wassermann test was negative. Roentgenographic examination of the chest was suggested but the patient refused.

The proposed treatment was to remove completely the right half of the mandible. The patient refused treatment and has not been seen since January 10, 1949.

COMMENTS—It is imperative that early diagnosis of all suspicious lesions be made. In this case the roentgenograms were not typical of a sarcoma. In obtaining the biopsy, the cortical plate was hard bone, but the medullary portion could be cut with a scalpel. Karsner[2] states, "Chondrosarcoma is likely to be central rather than peripheral, extends through marrow cavity and through bone cortex. Curable by early amputation, nevertheless it may metastasize and produce death."

2. Karsner, H. T., *Human Pathology*, ed. 6. Philadelphia: J. B. Lippincott Company, 1942.

UMI

Myxoma of the maxilla: *report of case*

■ *Ralph W. Edwards, D.D.S., Kansas City, Mo.*

The myxomata are tumors composed of embryonic connective tissue such as that found in the umbilical cord. They tend to recur unless thoroughly excised, and they can become malignant. Their occurrence in the jaw can be considered as unusual.

HISTORY—A Negro woman, aged 30 years, entered the University of Kansas Hospital on July 16, 1946, with a chief complaint of a swelling of the left maxilla. The swelling had been present for about three years.

CLINICAL EXAMINATION—Oral examination revealed a mouth with the dentition virtually wrecked from extensive caries. The right maxilla, posteriorly from the first incisor, was edentulous. In the left maxilla the second incisor was missing. An extremely hard tumefaction extended from the first incisor to the first bicuspid and superiorly well into the canine fossa (Fig. 1, left). The region was not painful.

ROENTGENOGRAPHIC EXAMINATION — An intraoral examination of the left maxilla revealed a region of destruction within the jaw extending from the midline to the first molar tooth (Fig. 2, left).

DIAGNOSIS—Cyst of the maxilla.

OPERATION—On July 18, after premedication of morphine sulfate grain 1/6 and atropine grain 1/150, anesthesia was induced by intravenous thiopental sodium. The remaining maxillary incisors, the left cuspid and first bicuspid were removed.

An incision was made along the alveolar ridge from the right second incisor region to the left first molar, with lateral incisions to facilitate the retraction of tissue. The mucosa covering the jaw in this region was retracted, and the alveolar process overlying the cyst was resected.

However, instead of the usual cystic contents, a type of tissue was encountered that had a rubbery consistency and a glistening white sheen. This tissue was removed in several sec-

School of Medicine, University of Kansas, Kansas City, Kan.

Fig. 1.—Left: Appearance of tumor of the left maxilla on July 16, 1946. Right: Appearance of the jaw on March 22, 1948

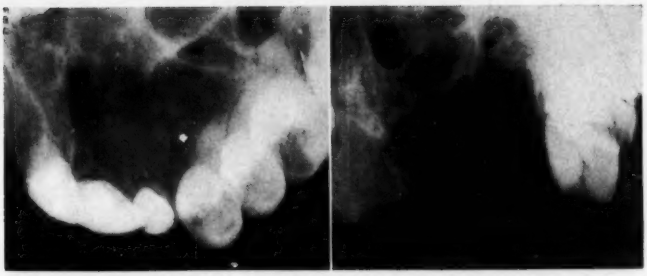

Fig. 2.—Left: Roentgenogram of left maxilla made on July 16, 1946, showing extent of destruction of bone by the tumor. A mistaken diagnosis of cyst was made. Right: Roentgenogram of left maxilla taken on March 22, 1948, showing regeneration of bone

tions from the bony crypt, the region was débrided, loosely packed with 5 per cent iodoform gauze and the mucosa closed with silk sutures.

PATHOLOGIC REPORT—Because of the unusual type of tissue encountered in this jaw, a rapid section examination was requested and reported the same day of the operation. Grossly, the tissue fragments were very irregular with numerous fibrous tags. On section the tissue for the most part consisted of soft, rubbery and homogeneous white material.

Histologically, the section was covered on one side by a typical layer of pavement epithelium with considerable infiltration of mononuclear leukocytes just underneath the epidermis. The underlying connective tissue appeared

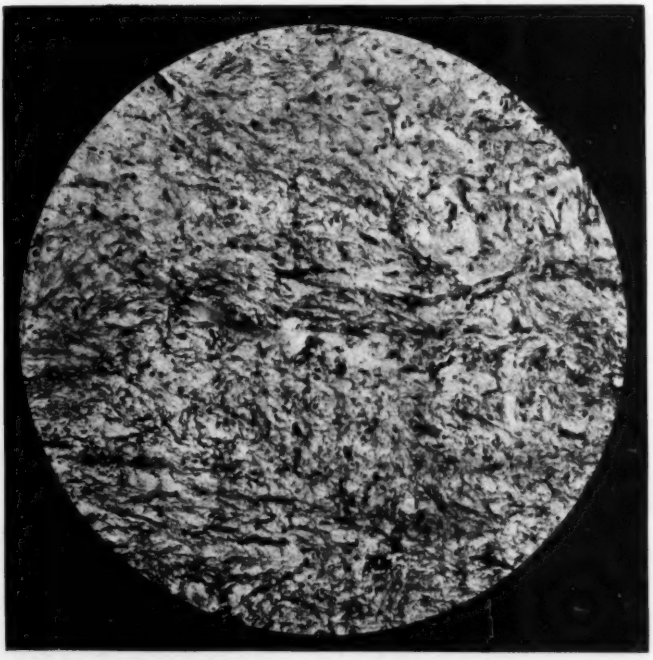

Fig. 3.—The tissue consisted of edematous appearing stroma containing irregular branching cells and showing cystic areas of degeneration and hemorrhages

to be unusually loose and edematous; in fact, the bulk of the tissue was made up of a loose, edematous appearing stroma containing numerous, irregular long branching cells and showing secondary cystic areas of degeneration and even hemorrhages. The picture suggested some type of myxoma. Some of the ground substance of the stroma showed a basophilic staining reaction. A few spicules of bone could be recognized here and there.

DIAGNOSIS—Myxoma of the jaw (Fig. 3). While there was no malignancy, the fact that this was myxomatous tissue makes recurrence quite possible.

COURSE—The postoperative course was without distress, and immediate treatment consisted of codeine grain ½ for the relief of pain. Hot saline irrigations were given orally every two hours during the day. A slight edema occurred in the region of the operation but slowly subsided. The gauze was removed from the jaw, and the patient was dismissed from the hospital on July 20, 1946. On July 27 the patient returned and the sutures were removed.

A roentgenogram made on March 22, 1948, showed regeneration of bone in the region of the tumor (Fig. 2, right). Figure 1, right, shows the appearance of the jaw on March 22, 1948.

ROENTGENOTHERAPY—Although it is recognized generally by roentgenologists that the myxomata are radioresistant tumors, it was considered advisable, because of the tendency of these tumors to recur, to prescribe roentgenotherapy. Consequently, 370 roentgens were given at the site of the operation on each of the following dates for a total of 1,850 roentgens: July 19, 20, 22, 26 and 29, 1946.

COMMENT—A type of tumor has been discussed that occurs infrequently in the jaws, and because of this fact its identity easily may be overlooked. In this patient an inaccurate roentgenographic diagnosis was made which was not discovered until the jaw was opened during the operation.—*300 West Forty-Seventh Street.*

Parotid calculi with mistaken diagnosis of residual roots: *report of case*

■ *C. Jay Brown, Jr., B.S., D.D.S., Kansas City, Kan.*

The following report deals with a case of misdiagnosis, substantiated by a misleading roentgenogram.

HISTORY—On October 1, 1943, a white man, aged 50 years, consulted his physician for relief from a badly swollen face. The patient had been bothered periodically with swelling and pain in the left side of his face. However, it had passed away without causing him enough alarm to seek medical advice. On the last occurrence swelling and pain were acute.

EXAMINATION—On examining the man, the physician obtained the accompanying roentgenogram. In the ramus of the mandible there appeared to be a radiolucent area. Inside this area were two structures similar to the roots of teeth in their radiopacity. The patient remembered having an impacted third molar removed some years before, so it was assumed that the mesial and distal roots were still present.

The patient was referred to this office for removal of the retained roots. Intraoral roentgenograms did not show any areas of infection or any retained roots in the region.

In retracting the cheek to examine the mouth, pus was expressed from Stenson's duct. A small dental film was placed in the oral ves-

Extraoral roentgenogram showing radiopaque objects that resemble lower molar roots in left ramus of the mandible

tibule and the rays were directed toward the parotid region. This film showed the calcifications in the parotid duct.

TREATMENT—A small silver probe was passed through the duct to permit drainage. The calcifications could be felt and moved with the probe. Drainage was continued in this manner for five days; at this time the patient's swelling and pain had subsided.

While manipulating the calculi to facilitate drainage, a sharper probe was used, and a small piece of stone was broken off. This piece easily was worked out of the duct. Further attempts were made at subsequent appointments, and each time it was possible to reduce the size of the obstruction. Eventually the entire stone was removed in this manner.

COURSE—At the present writing, there has been no recurrence of calculi in this patient. It is extremely rare that salivary stones can be removed without surgery. The soft structure of this stone facilitated its fracture and removal. The case clearly shows how easily this condition may be confused with other disorders, and how misleading a roentgenogram may be.

CONCLUSION—In the diagnosis of swelling about the face and neck, salivary obstruction is at the bottom of the list. This complication commonly is confused with acute alveolar abscesses, cellulitis, maxillary sinusitis, parotitis and other diseases.

Of the salivary glands to be involved with calculi, the parotid gland is the least common.[1]

Often it is the unfortunate habit of human beings to examine an object closely and see not what is actually present, but to alter the circumstances to meet a suspected diagnosis. Often one sees what one expects to find, instead of what is really present.—*3214 Strong Avenue.*

1. New, G. B., and Harper, F. R., Chronic Inflammation of the Salivary Glands With or Without Calculi. *Surg., Gynec. & Obst.* 53:456. 1931.

Oral cancer—the surgeon's responsibility

APRIL HAS BEEN designated as cancer month. It is the month during which the public and the healing arts professions are presented with information to make them more cancer conscious. This is a very commendable program but to the alert conscientious oral surgeon every month is cancer month—every day is cancer day.

The problem of cancer is ever before us—continually in our thoughts. We must consider cancer not only from the standpoint of diagnosis and treatment but from the standpoint of prevention. Cancer is a matter of national interest because of its 170,000 deaths and its 680,000 casualties annually. Neither war nor famine so consistently is responsible for the economic waste of human resources and the great suffering incident to this disease.

Early cancer and many advanced cancers are curable. At one stage all cancers consist of a small group of cells clinically insignificant. At this stage its eradication from the local region is a curative measure. Unfortunately, in some regions, the malignant growth does not make itself evident until an advanced stage is reached. Generally this is not true of oral cancer.

Although early mouth cancer seldom causes distressing symptoms its presence in an easily accessible, highly sensitive region should command immediate attention from the patient and the dentist. In no other part of the body is such an opportunity offered for early detection.

Because of the close proximity of the lesions to the teeth many patients logically first consult with their dentist. Dr. Hayes Martin points out that 60 per cent of all patients, in a large series, with cancer of the gingiva consulted a dentist first. From 20 to 39 per cent of the patients with cancer in other parts of the mouth first consulted a dentist. Many of these patients owe their lives to the alert observations and early attentions of the dentist who made the diagnosis or referred them to a physician for diagnosis and treatment. Martin's study indicated that in spite of early consultation with a dentist many malignant lesions went unrecognized or were incorrectly diagnosed. In some cases ineffectual dental remedies were employed. Despite early recognition of cancer by the dentist there was a delay of three and a half weeks between the first visit to the dentist and the first consultation with a physician

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for treatment. In the unrecognized cases seen by a dentist there was an average delay of eight and a half weeks. This delay in many instances was due to the noncommittal attitude of the dentist or his reassurance to the patient that the condition was not serious. Delay of even a few days may determine the possibility of cure.

Patients with any suspicious lesions should be given every possible consideration. In all patients more than 40 years of age the oral surgeon should think first of cancer. In all patients less than 40 he should think of cancer too. Cancer of the mouth, because of the ease of clinical inspection and palpation, is not difficult to detect. Both patient and surgeon should be aware of any abnormal condition soon after its appearance. Biopsy of any oral area is simply and conveniently done under local anesthesia as an office procedure. If a malignant lesion is present treatment should be instituted at once. If premalignant lesions are noted eradication or close observation is indicated. The management of other lesions varies with their character.

In general the treatment of oral cancer, except in the hands of a few highly trained individuals, is not a problem for the oral surgeon. The oral surgeon can however, in many cases, be of invaluable aid to the medical profession in the management of oral cancer. The removal or correction of areas of chronic irritation, infection, and carious teeth should precede any form of treatment. Good oral care is imperative.

If the treatment involves the use of irradiation lead screen appliances should be constructed for the protection of uninvolved tissues. If the irradiation must involve bone in which teeth are present all of the teeth, regardless of their condition, must be extracted and the soft tissue over the alveolus be permitted to heal before treatment is undertaken. Irradiation of segments of the jaws containing teeth predisposes to disintegration of tooth structure, irradiation necrosis or osteomyelitis of the partially devitalized bone.

A word of caution regarding the extraction of teeth in previously irradiated areas is in order. Quiescent areas irradiated years previously may be opened to uncontrolled infection by a simple extraction. Conservatism in management of this type of case may save the patient extreme suffering and the surgeon considerable embarrassment.

The following rules should be kept in mind regarding all oral lesions: (1) have all abnormal tissues removed from the mouth examined by a competent pathologist; (2) in all patients more than 40 years of age consider the lesion cancerous until it is proved otherwise; (3) in patients less than 40 consider cancer in the differential diagnosis; (4) excise and examine microscopically all small suspicious lesions; (5) obtain a specimen of all large suspicious lesions for biopsy; (6) extract all teeth from regions to receive intensive irradiation treatment; (7) exercise extreme caution in the surgical treatment of any previously irradiated bony area.—*Reed O. Dingman.*

■ Persistent Chronic Dislocation of the Mandible

Q.—I have a patient 38 years old who has a permanent anterior bilateral mandibular dislocation of 9 years' duration. This was a spontaneous dislocation which was never reduced. He has a protrusive bite relationship and roentgenograms show the condyle heads dislocated anterior to the articular eminence of the temporal bone. Opening and closing movements are possible but lateral and protrusive motion is impossible.

One surgeon suggests resection of the condyle heads; another thinks bilateral horizontal section of the ascending ramus would permit repositioning of the anterior part of the mandible.

How should this case be managed?—*Howard Lawrence, M.D., Columbus, Ohio.*

A.—To our knowledge, no case of this duration has been reported in the literature. One of our cases of 9 months' duration was successfully partially reduced under general anesthesia. Stout rubber band traction posteriorly, directed over a period of one week, gave final satisfactory permanent reduction.

Resection of the condyles or transverse osteotomy should not be necessary and would not give satisfactory results.

The first method should be an attempt to reduce the dislocation forcibly under general anesthesia. This probably will not be completely possible, but probably some degree of reduction will be possible. This should be followed by strong rubber band traction posteriorly by means of upper and lower arch bar attachments. Instructions should be given the patient to manipulate frequently the mandible downward and posteriorly by biting on a small solid object with upward pressure applied simultaneously to the symphysis.

Over a period of a few weeks this method should give satisfactory results. If not, open reduction is indicated.

■ Surgical Treatment of the Diabetic Patient

Q.—Can a patient suffering from diabetes be prepared safely to undergo oral surgery?—*L. E. McK.*

A.—Usually the patient's physician can initiate an insulin regime and diet control which will make a surgical procedure safe. However, many times surgical procedure will cause a postoperative change in the insulin requirements which may persist until recovery is complete. For this reason, it is of the utmost importance to have the urine checked for sugar at frequent intervals, postoperatively, that any change in insulin requirement may be effected. Usually, daily checks are sufficient until recovery is complete.

■ Handling of Postextraction Complication

Q.—When granulomas are encountered in extraction sites, is it necessary to remove all of the granulomatous material?—*C. K. M., D.D.S., Ann Arbor, Mich.*

A.—Theoretically it is the best surgical procedure to curet completely all questionable granulomatous tissue from the operative field. However, roentgenographic and clinical studies have demonstrated that if reasonable attempts are made to remove granulomatous tissue and the vascularity is reestablished, normal healing process should ensue. This does not hold true in cyst formation where excochleation of the limiting membrane should be complete, or prepared by Partsch procedure to facilitate healing by second intention.

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■ Flaps

Q.—What are the general principles to be considered in the design of a flap for a surgical procedure?—*Horstkotte, D.D.S.*

A.—The principles of flap procedure are:

1. The flap must have an adequate blood supply. The base should be at least as wide as the free margin. Avoid sharp acute angles on the flap margins.
2. Include the entire thickness of the mucoperiosteum.
3. Where teeth are present start vertical incisions in interproximal areas to minimize trauma and facilitate gingival reattachment.
4. When a vertical incision is to be used make it mesial to the surgical area and one tooth distant.
5. Make the flap sufficiently large so that the incised margins are supported on solid bone when the flap is sutured into place.
6. Adequate access often may be obtained by elevation of the mucoperiosteum from the gingival margin for a distance of one or two teeth on either side of the operative site. This will permit retraction of the tissue and avoid making a vertical incision, which is definitely a postoperative advantage.

■ Determination of Bleeding and Clotting Time

Q.—What practical office procedure can be done to determine fairly accurately the bleeding and clotting time when such information is necessary?—*P. M. N., Ann Arbor, Mich.*

A.—For ordinary office routine, bleeding and coagulation time may be determined simultaneously from a skin puncture (a no. 11 Baird-Parker blade or a blood lancet is suitable) deep enough to insure a free flow of blood from either the finger tip or the ear lobe. Do not force the flow of blood.

Bleeding time is determined by collecting the drops of blood from the puncture wound on absorbent paper at half minute intervals, without allowing the paper to touch the skin. Normally, the drops will become progressively smaller and usually will cease forming in one to three minutes. The maximum time which may be considered normal is eight minutes.

There are two simple methods for determining coagulation time, using blood from the same puncture wound used for the bleeding time. Collect several drops of blood on a clean glass slide, and at one minute intervals draw a needle through a drop. When a fine fibrin thread clings to the needle, coagulation has taken place.

The other method is to take up free-flowing blood from the puncture wound in a clean capillary glass tube about 1.5 mm. in diameter. At one minute intervals carefully break short sections from the tube. When a thread of fibrin spans the broken sections, coagulation has occurred. The normal time for both these methods is two to five minutes.

For a more accurate means of determining coagulation time, the Lee-White method should be used. Blood is secured from a vein, using a small syringe and avoiding suction. Record the time blood first appears in the syringe. Place 1 cc. of blood in a test tube 8 mm. in diameter which has been rinsed with physiologic saline solution. Tilt the tube at one minute intervals, and when the tube can be inverted without displacing the clot, coagulation is complete. The normal time for this method is five to ten minutes.—*Verna Harner, assistant bacteriologist, School of Dentistry, University of Michigan.*

■ The Problem of Acutely Infected Teeth

Q.—Should teeth be extracted in instances of acute exacerbation when swelling and other inflammatory signs exist?—*C. K. M.*

A.—Since the advent of chemotherapy and antibiotics it is now much safer to extract teeth in the presence of an acute infection than previously. This should be done, however, only when it is believed that the extraction of the tooth will facilitate drainage. If the infection has localized in adjacent areas where incision and drainage are indicated and if there is likely to be difficulty in removal of the tooth, the ex-

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traction should be deferred until after the acute inflammation has subsided. Extensive surgical procedures for tooth removal may spread the infection and should be deferred until the acute process subsides. If it seems necessary to extract the tooth the patient may be premedicated with penicillin, 300,000 units given one hour preoperatively. A close follow-up is indicated, and antibiotics should be administered twenty-four to forty-eight hours after the acute phase subsides.

■ Methods of Obtaining Biopsy Specimens

Q.—What are the best methods of obtaining a specimen for biopsy, and how should the specimen be treated for transportation to the pathologist?—*Hopkins, D.D.S.*

A.—Incision by sharp scalpel is usually the best method. However, there are other methods that may prove advantageous in special cases. They are: (1) incision by (a) scalpel, (b) electrocautery and (c) high frequency cutting current; (2) punch biopsy; (3) curettage; (4) paracentesis for examination of fluids for tumor cells; (5) aspiration.

The specimen should be dropped in a bottle containing a 10 per cent formaldehyde solution twenty times the volume of the specimen. Never put the specimen on dry cloth or in an empty bottle and add the solution later.

■ Accidental Sinus Opening

Q.—Recently I removed a maxillary second bicuspid. I suspected the apex of this tooth communicated with the maxillary sinus. What method could I use to evaluate this at the time of operation?—*A. L. Lasky, D.D.S., Ann Arbor, Mich.*

A.—Close off the external nares and instruct the patient to attempt blowing gently through the nose. A bubbling of the blood from the alveolus indicates a communication; if there are no bubbles you can be reasonably certain there is no penetration of the sinus lining.

The answers here published are prepared by competent authorities, but are not to be construed as the opinions of the editorial board, or of any official body. Anonymous notes and postcard queries will not receive consideration. The writer's name and address will be omitted on request, but it must be contained in the letter. Communications must be addressed to the editor.

■ Honor Past-President of A. D. A.

A two-day testimonial program was held in Washington, D. C., March 11 to 12, in honor of Oren A. Oliver, past-president of the A.D.A. Dr. Oliver is professor of clinical dental surgery and chief visiting surgeon, Vanderbilt University School of Medicine and editor of the *American Journal of Orthodontics and Oral Surgery*.

The program was planned by Walter T. McFall, Asheville, N. C.; Leigh C. Fairbank, Washington, D. C.; E. C. Lunsford, Miami, Fla.; Frank P. Bowyer, Knoxville, Tenn., and Boyd W. Tarpley, Birmingham, Ala.

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■ THE PATHOLOGY OF TUMOURS. *By R. A. Willis. 991 pages, 500 illustrations. Price $20. St. Louis: C. V. Mosby Co., 1948.*

This excellent work is the result of Willis' interest in tumors based upon long experience as a hospital pathologist and many years devoted to research. This work is primarily a personal record of his own observations and conclusions. It is refreshing to note that the author avoids the noncommittal vagueness on controversial issues often noted in similar works. On any controversial subject he states the various prevalent theories, and then clearly presents his own views on disputed matters. This text is considered by many to be the most important and convenient reference available for reliable information on the pathologic aspects of individual tumors.

Part I, 207 pages, containing the first twelve chapters, deals with the definition of tumors, their classification, growth and direct spread, metastasis and experimental production. The author presents a very comprehensive definition of a tumor in which he can rule out the inflammatory and reparative growths and malformations which are considered by some to be neoplasms. Willis discusses the classification of tumors completely. He prefers a basic histogenic classification, avoiding the erection of artificial subdivisions. His classification is simple and precise, and he deplores many of the long, compound, descriptive names often found in the literature. The question of the innocent or malignant condition is well covered in detail.

Part II, 784 pages, fifty chapters, includes the complete description, pathology, and clinical aspects of the various tumors in different parts of the body. The oral surgeon will find much of interest in these chapters as nearly any tumor may occur in the mouth. Epithelial tumors are comprehensively covered in chapters 13 and 14. Chapter 15 is devoted to carcinoma of the lips, mouth, pharynx and larynx. Chapter 16 is of interest to the oral surgeon, being devoted to tumors derived from the epithelium of the lamina. Willis believes that there has been a needless elaboration of the classification and naming of the malformations, cysts and tumors of the tooth connective tissues. He believes the only essential subdivisions of these lesions are: (a) adamantinoma, (b) intra-alveolar epidermoid carcinoma, (c) odontomes, and (d) epithelium-lined cysts. The adamantinomas and the rare intra-alveolar carcinoma are well described. Willis considers that the terms adamantinoma or ameloblastoma are both erroneous in that the internal enamel epithelium, or layer of ameloblasts, does not produce this tumor. The typical marginal columnar cells and central areas of stellate reticulum may be present in one part of a tumor, whereas in other parts the cells may closely resemble basal cell carcinoma of the skin, especially of the cystic type. These tumors distend, invade and destroy bone, and tend to recur. Attempts to distinguish benign and malignant adamantinomas are futile. Surgical intervention must be thorough. Willis would call these growths "carcinomas of the tooth germ residues." He believes that the odontomes and cysts are malformations or inflammatory growths, and he gives little space to their consideration. Epithelial tumors of the salivary glands are completely covered in Chapter 17. Considerable space is given to the discussion of the so-called "mixed" tumors which Willis believes are all pleomorphic adenomas or adenocarcinomas. He believes that the term "mixed" tumor is a misnomer and should not be used. These tumors are derived from the epithelium of the salivary glands and are not of extrinsic origin, such as branchial inclusions. He states they "are far from benign, showing all gradations of structure and malignant behavior."

Chapter 18 contains a discussion of the difficulty of exact pathologic diagnosis of tumors of the maxilla and maxillary sinuses on account of the resemblance of adamantinomas (carcinoma of tooth germ residues), basal cell carcinomas, adenocarcinomas and pleomorphic

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salivary (mixed) tumors. Serial sections from all portions of the tumor should be examined for stellate reticulum and other adamantinomatous epithelium. These tumors, though of slow growth, are difficult to eradicate and are usually fatal, but they rarely metastasize.

The oral surgeon will also find the discussion of mesenchymal tumors (Chapter 42) and tumors of cartilage and bone (Chapter 43) very informative. The section of Chapter 43 on osteoclastoma (or giant cell tumor of bone) is very interesting in that Willis' opinions are at variance to those held by many American pathologists who consider these growths inflammatory rather than neoplastic. Willis believes that the giant cell epulis seen in the mouth is a granuloma and not a neoplasm. His conclusions on the "Ewing's tumor" are worthy of consideration. He believes "Ewing's tumor" is not a pathologic entity but rather an ill-defined syndrome of a nonosteogenic round cell radiosensitive bone tumor usually seen in young persons. The syndrome can be caused by several different tumors. He believes most of the reported cases of "Ewing's tumor" are metastatic growths of neuroblastoma or occasionally a primary reticulum cell sarcoma.

The tumors of vascular and lymphoid tissue (Chapter 49) receive considerable attention. The author believes lymphosarcoma and lymphatic leukemia are not separate entities but only variants of a single disease process.

While this work was written primarily for pathologists, the practicing oral surgeon will find much of interest in it. The author writes in a very clear and forthright style which is to be commended. The photomicrographs are well selected and clearly reproduced. A valuable list of select references is placed at the end of each chapter with frequent comments by the author. The index is complete. The text is a comprehensive, soundly critical addition to the literature on tumors.—*C. W. Waldron.*

■ PSYCHODYNAMICS AND THE ALLERGIC PATIENT. *By Harold A. Abramson, M.D., F.A.C.S. 81 pages, 7 illustrations. Price $2.50. St. Paul, Minn.: The Bruce Publishing Co., 1948.*

This book covers the material given at the panel discussion on psychiatry and allergy at the third annual meeting of the American College of Allergists, held in Atlantic City, New Jersey, June 8, 1947. It is written in cooperation with ten panel discussers and includes the remarks made from the floor by seven members of the Association. The biographies are confined to basic material. Since the panel expresses the view of dermatologist, psychiatrist, psychologist and allergist, it should be of primary interest to all of these groups as well as the general practitioner.

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■ Surgical Repair of Lacerations and Fistulas of the Parotid Duct. *Hugh A. Bailey and Victor Skaff.* Ann Surg. 129:103, January, 1949.

A series of 5 cases in which closure of a fistula of the parotid duct was effected is reported. Previous methods of closure reported in the literature are reviewed and a modification of the "dowel" technic is described.

Under local anesthesia a skin incision is made to expose the site of injury. A probe is then passed from the mouth through the duct to the incision. Number 10 cotton or heavy silk is attached to the probe and drawn into the mouth. Next, the severed end of the duct proximal to the gland is identified, using lemon juice to stimulate salivation if necessary. Then, using the blunt end of a round needle, the suture is passed back through the duct into the gland and continued to the skin. The skin is nicked and the suture is picked up. A lead shot is attached to the suture at both ends, a small square of rubber dam being placed between the shot and oral mucosa to minimize irritation. The tissue in the incision is sutured with catgut and the skin is closed without drainage. A watery discharge from the operative site may be expected to persist for about seven days. The skin sutures are removed in about seven days, and the suture maintaining patency of the duct can be removed in approximately fourteen days.

In the cases reported, function of the duct, evidenced by the appearance of saliva in the oral cavity, began in a few days. The shortest interval was a few hours, the longest, seven days. In different cases the "dowel" suture was maintained from twelve to twenty-four days. None of the cases reported had a history of a fistula present for more than thirty days.

The authors suggested the following classification of fistulas according to location: (1) over the gland; (2) over the masseter muscle; (3) in the buccinator area. In their opinion, if roentgen therapy is employed in lacerations of the gland and accessory ducts, surgical intervention will not be required in many cases. They do not recommend short circuiting operation to repair fistulas originating in the buccinator region, because the valvelike action of the papilla is lost leaving the duct vulnerable to retrograde infection. Cases of this type were included in the series and the technic described gave good results.—*John W. Hamilton.*

■ Oxygen Therapy in Surgery. *Wendell H. Kisner.* Am. J. Surg. 76:76, July, 1948.

This article discusses the therapeutic value of oxygen in surgery. It reviews the respiratory physiology with emphasis on oxygen lack and carbon dioxide excess.

The widespread use of morphine, scopolamine and barbiturates—all of which depress the respiratory center—must be kept in mind in considering oxygen therapy.

Head injuries, shock, anesthesia and pulmonary complications are the conditions most benefited by oxygen.

Seven per cent carbon dioxide and 93 per cent oxygen is of greater value than pure oxygen alone, because from 5 to 7 per cent carbon dioxide with oxygen increases cerebral blood flow up to an average of 75 per cent, and it also increases pulmonary ventilation.

Cyanosis and dyspnea are not present in the early stages of anoxia. More use of 7 per cent carbon dioxide and 93 per cent oxygen mixture as a preventive and therapeutic aid for anoxia should be seen.—*C. Litton.*

■ Cancer of the Lip. *Ian MacDonald, M.D.* California Med. 68:37, January, 1948.

There is no valid reason not to reduce mortality of cancer of the lip to a bare minimum. The facts that the majority of these lesions

develop over a long period of time and respond readily to preventive or curative treatment are of great aid in stopping cancer before it reaches the destructive or noncurable stage. Cancer of the lip develops very slowly and almost invariably on the exposed borders of the lip where it can easily be seen by the physician or dentist, making it easily accessible for diagnosis or treatment.

Any lesion on the lips, particularly of males past middle age, should be considered cancerous until proven otherwise. With this in mind, many cancers of the lip could be prevented or stopped in their precancerous stage.

To speak of precancerous lesions does not mean that cancer will inevitably follow, but the chances are 50 per cent or higher that cancer will develop at the site of the lesion. The most common precancerous lesions are keratosis and leukoplakia.

Causative factors involved are, thermal or chemical irritants, long and repeated exposure to sun rays and wind and repeated trauma. All of these may be classed as chronic irritants. Another possible factor, though not proved important, is vitamin deficiency.

Early excision of any lip lesions of leukoplakia or keratotic nature showing erosive or ulcerative tendency is possibly the best method of treatment. There are other methods of treatment such as brush cautery, electrocoagulation or actual deep cautery. However, these methods do not offer the distinct advantage of biopsy study to confirm the previous diagnosis. Irradiation in most of these cases is not indicated.

Patients with dry lips that crack or fissure easily should take precautionary measures, particularly if previous lesions have been removed. Regular use of a wool fat ointment as protection for the lips has shown good response. Large daily doses of brewer's yeast as a source of vitamin B complex is of some value. Patients who are sensitive to sun rays should avoid exposure if possible. Chronic irritants of any nature, particularly tobacco, should be eliminated.

Sites of precancerous lesions or of chronic irritations are the usual sites of origin of squamous cell carcinoma. However, in some cases carcinoma may develop in apparently healthy tissue.

Carcinoma of the lip involves the lower lip in 96 per cent of the cases, the upper lip and commissures in 4 per cent of the cases. A greater per cent (80 per cent) occurs in males, usually past the age of middle life. Young adults, however, are not immune.

Carcinoma may begin in a papillary or mild ulcerative form. The papillary type may present a thick, raised plaque or scaly type wart on the mucosa. Its progress is slow but finally breaks down, undergoes necrosis and infiltration. The ulcerative form is faster with early destruction and infiltration seen. In either case secondary infection, increased pain, edema and cellulitis may be resultant complications.

While cancer of the lip is considered a deadly disease, it is curable if properly treated in the early stages. Ninety per cent of untreated, uncomplicated cancers of the lip are definitely considered curable. The first treatment should be adequate since only 40 to 60 per cent of recurrent lip cancers are curable.—*C. E. Hopkins.*

■ THE USE OF VITALLIUM PLATES TO MAINTAIN FUNCTION FOLLOWING RESECTION OF THE MANDIBLE. *B. S. Freeman.* Plast. Reconstr. Surg. 3:73, 1948.

Resection of the mandible necessitates immediate postoperative fixation of fragments to prevent external and internal distortion resulting in respiratory distress, feeding problems and severe deformity. In adults with teeth retention of the proper relationship between fragments, with the later restoration of bone, continuity can be accomplished with intraoral wires or dental splints. In edentulous adults and children, retention of the fragments is difficult. Immediate bone grafting, in our experience, has failed because of either oral contamination or incomplete fixation of the fragments. Following resection of a sizable portion of the mandible without stabilization, the mental anguish and depression, although great, are secondary to the immediate serious difficulty in respira-

tion, mastication and speech. Vitallium plates, fixing both fragments firmly in their original places and maintaining rigidity of the mandible during the period between operations, have been successfully used to prevent these sequelae.

Simple Sherman type plates of vitallium, in varying sizes, can be bent to proper curvature in the operating room. If necessary, a specially curved plate can be prepared in any dental laboratory equipped to make vitallium prostheses. The area to be resected is outlined; the metal plate is bent to the approximate desired curvature with a pair of pliers or the fingers; the positions of the plate holes are marked with drill holes made into the bony part to be left. After the oral cavity has been closed tightly, the wound irrigated and the instruments and gloves changed, the plate is attached by small vitallium screws or bolts.

Even in the presence of wound infection, metal plates can be allowed to remain in position for a considerable period of time in order to stabilize the bone fragments and prepare them, if necessary, for a later graft.—Louis T. Byars. *Surg., Gynec. & Obst. (Internat. Abstr. Surg.)* 87:19 (July) 1948.

■ AN INVESTIGATION OF GELATIN SPONGE WITH THROMBIN AND PENICILLIN IN THE TREATMENT OF ORAL SURGICAL WOUNDS. *Lester M. Silverman, D.D.S., M.S.D.* Oral Surg., Oral Med. & Oral Path. 2:260, February, 1949.

Gelatin sponge was used in combination with penicillin and thrombin in an effort to effect bacteriostasis, hemostasis and clot support. Gelatin sponge is one of a group of new materials developed and investigated extensively in recent years. This group also includes fibrin foam, oxidized cellulose, and starch sponge. They all have the common property of hemostasis and absorbability. A review of the literature indicates that oxidized cellulose has a tendency to expansion after absorption of blood, followed by extrusion of the clot and resultant breakdown. Starch sponge has the disadvantage of being friable and impractical for surgical wounds. There appears to be no difference in tissue reaction to fibrin foam and gelatin sponge. However, the gelatin sponge is considered an excellent nonantigenic material since its essential component, gelatin, is a universal protein deficient in the amino acids containing aromatic radicals. The anaphylactic and precipitin reactions depend on these radicals.

In this study 105 cases of multiple extractions were used in such a way that experimental and control sockets could be compared under similar conditions. Eighty-five of the experimental and control cases were bilateral extractions of teeth in the same general areas of the mouth, molar versus molar, premolar versus premolar. A 1 cc. saline solution of thrombin containing 200 units of thrombin per cubic centimeter was mixed with 200,000 units of crystalline penicillin G sodium under sterile conditions. The gelatin sponge was cut to approximately two-thirds the size of the tooth socket and was placed in the container of thrombin penicillin solution. Air was expressed from the meshes of the material with forceps, and the sponge soaked up the solution and resumed its size and shape. Immediately after the extraction of the tooth the socket to be implanted was sponged dry and the gelatin sponge placed well in the socket where it filled with blood and swelled to the volume of the socket. Where necessary, a suture was placed across the surface of the socket to maintain the sponge.

The control sockets were handled as routine extractions and observed for postoperative hemorrhage for five minutes. Observations were made postoperatively at twenty-four hours, forty-eight hours, seventy-two hours, and at one week for hemorrhage, pain and healing. Postoperative pain was determined by inflammatory reactions and subjective symptoms of the patients. Healing was evaluated by the nature of the clot and the extent of epithelization. Sections were removed from the healing experimental and control sockets for histologic study. Serum levels of penicillin were made at one, three, four, five and six hour intervals following implantation of the gelatin sponge soaked in thrombin penicillin solution.

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Statistical analysis indicated a highly significant decrease in postoperative hemorrhage with the use of gelatin sponge impregnated with thrombin and penicillin. There was also reduction in the incidence of postoperative pain. This was attributed to several factors. The penicillin acting locally as a bacteriostatic agent prevented clot breakdown due to bacteriolysis. The sponge acted as a mechanical barrier to food and debris which normally accumulate in postextraction wounds, and as irritants initiate an inflammatory reaction. There was also better healing in the experimental group of sockets.

At one hour the serum penicillin levels averaged 0.85 units per cubic centimeter, decreased to 0.12 at the end of four hours, and to 0.01 at six hours.

Histologic study showed that the sponge acted as a support for the clot and as a scaffold for new capillary formation. Disappearance of the gelatin sponge from the socket was brought about in an average of twenty-five to thirty days by the phagocytic action of giant cells. The sponge as a foreign material did not impede organization and repair of the socket. The rate, manner, extent of repair, osteoblastic and osteoclastic activity were comparable to the untreated extraction sockets.—*E. H. Horstkotte.*

■ Reactions to Penicillin. *Samuel I. Kaplan, D.D.S. and George Hurwitz, M.D.* Oral Surg., Oral Med. & Oral Path. 2:21, January, 1949.

Since the advent of penicillin, the treatment of dental infections has been simplified. With the increased use of this antibiotic, however, there has been a parallel increase in the number of reactions associated with its administration. Although most of the reactions are of minor significance, nevertheless, many are severe and are the cause of concern to the patient as well as to the individual administering the drug.

The authors gave 403 injections of penicillin, of which 203 were given with the preparation of penicillin in beeswax and oil, and 200 with procaine penicillin. Two types of reactions occurred in conjunction with these injections. One type of reaction occurred only in those patients receiving penicillin in beeswax and oil. There were fourteen such reactions. The other type of reaction was an allergic one to penicillin and occurred in both types of preparations. Four of these reactions occurred in a total of 200 injections.

The authors discussed the four possible types of reactions that may follow the administration of penicillin preparations. These were (1) inflammatory reactions due to the foreign body effect of the medium in which the penicillin is dispersed; (2) idiosyncrasy to the procaine in procaine penicillin preparations; (3) allergy to procaine; (4) allergic reaction to penicillin itself or associated impurities.

The first reaction is associated only with the beeswax and oil preparations and is more common in females than males.

The idiosyncrasy to the procaine is analogous to the mild central nervous system reaction occurring in some patients after the administration of procaine anesthetic.

The occurrence of allergic reactions to procaine is associated only with individuals having asthma or hay fever, in which case the administration of procaine penicillin may precipitate an attack of the allergy.

In the last type of reaction mentioned there is no relationship between the allergic reaction to penicillin and the presence of asthma or hay fever in the individual. The reaction resembles serum sickness, with the occurrence of such manifestations as urticaria, angioedema, fever, joint pains, swelling and enlargement of the lymph nodes.

In case of the development of a localized inflammatory reaction at the site of injection of penicillin in beeswax and oil, the authors suggested the application of hot moist compresses to the affected area.

Those patients who have an idiosyncrasy or are allergic to procaine should not receive procaine penicillin.

Allergic reaction to penicillin should be treated by the administration of antihistaminic drugs (50 to 100 mg. of "Pyribenzamine"

every four hours was suggested as adequate treatment). Included among other antihistaminic drugs are "Benadryl," epinephrine and procaine.—*De Orr Cooley.*

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■ REPORT OF THE COMMITTEE ON CERTIFICATION OF SPECIALISTS. *Earl W. Swinehart, chairman.* J. Am. Col. Den. 15:111, September, 1948.

The committee presents the results of a study made in the six states requiring the certification of specialists. Such laws have been enacted in Illinois, Oklahoma, Tennessee, Michigan, Kansas and South Carolina. Much of the material obtained was from questionnaires sent to three men from each state: an official of the state board of dental examiners; an official of the state dental society; a specialist.

Some of the information revealed by the questionnaires was as follows:

1. Seventeen consultants in the six states report that the specialists' law has had the desired result of protecting the public from the unprepared or self-styled specialists.

2. Sixteen consultants report that specialists' laws serve to improve the standards of special practices.

3. The board officials in all six states report that the requirements for certification by the proposed national specialty boards are generally higher than their state requirements.

4. The board officials in five states report that in the light of the proposed certification of specialists by national boards, they would not be satisfied to nullify the present specialists' clause of their respective dental acts.

5. Since the enactment of the specialists' laws in the six states 678 have been certified to practice as specialists in some branch of dentistry; 149 have either failed to meet the requirements of the boards or failed in their examinations. The ratio is 1 failed to every 4½ certified, or slightly over 22 per cent.

A partial list of the opinions set forth by the committee are:

1. As a result of the lengthening and the strengthening of the dental curriculum, dentists today have an improved educational background for specialization. However, these improved standards alone do not qualify a dentist as a specialist.

2. According to the survey there are many dentists seeking to specialize who are wholly unprepared to perform adequately the services of a specialist.

3. Only through state laws can these dentists be prevented from representing themselves as specialists.

4. State laws with their periodic raising of requirements are a positive force in elevating all levels of professional practices.

5. If special practices in dentistry are to reach their peak in knowledge and service, certification by state statute must have first consideration.

6. The operation of the specialists' laws has proved generally satisfactory to the public and the profession.

7. These laws have had the desired results in protecting the public from the unprepared, the misfit and the pseudo specialist.

The committee concluded that:

1. The principle and the purpose of the laws certifying specialists in dentistry have been upheld.

2. The experience of the six states having these laws supports the conclusions that the public should have the benefits and the protection of laws providing for legal certification of specialists, and that these laws have earned and deserve a rightful place in state dental acts.—*S. Elmer Bear.*

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